

P-Element Insertion Alleles of Essential Genes on the Third Chromosome of *Drosophila melanogaster*: Correlation of Physical and Cytogenetic Maps in Chromosomal Region 86E-87F

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ABSTRACT

We have established a collection of 2460 lethal or semi-lethal mutant lines using a procedure thought to insert single *P* elements into vital genes on the third chromosome of *Drosophila melanogaster*. More than 1200 randomly selected lines were examined by *in situ* hybridization and 90% found to contain single insertions at sites that mark 89% of all lettered subdivisions of the Bridges' map. A set of chromosomal deficiencies that collectively uncover ~25% of the euchromatin of chromosome 3 reveal lethal mutations in 468 lines corresponding to 145 complementation groups. We undertook a detailed analysis of the cytogenetic interval 86E-87F and identified 87 *P*-element-induced mutations falling into 38 complementation groups, 16 of which correspond to previously known genes. Twenty-one of these 38 complementation groups have at least one allele that has a *P*-element insertion at a position consistent with the cytogenetics of the locus. We have rescued *P* elements and flanking chromosomal sequences from the 86E-87F region in 35 lines with either lethal or genetically silent *P* insertions, and used these as probes to identify cosmids and P1 clones from the *Drosophila* genome projects. This has tied together the physical and genetic maps and has linked 44 previously identified cosmid contigs into seven "super-contigs" that span the interval. STS data for sequences flanking one side of the *P*-element insertions in 49 lines has identified insertions in the $\alpha\gamma$ element at 87C, two known transposable elements, and the open reading frames of seven putative single copy genes. These correspond to five known genes in this interval, and two genes identified by the homology of their predicted products to known proteins from other organisms.

TRANSPOSABLE elements are now widely used for mutagenesis as they couple the advantages of providing effective genetic lesions with the ease of detecting disrupted genes for the purpose of molecular cloning. Early attempts to utilize the *P* transposable element of *Drosophila* required the mobilization of large numbers of natural *P* elements (SPRADLING and RUBIN 1982; ENGELS 1984; KIDWELL 1987). Lines obtained in this way were difficult to use either for genetic or molecular studies since extensive out-crossing and recombination experiments were required to eliminate *P* elements other than the one responsible for the mutation of interest. Consequently the development of a means for mobilizing a single marked *P* element in each mutagenized strain was a considerable step forward

(COOLEY *et al.* 1988). The advantages of such "single *P* element" mutagenesis were further improved by the modification of *P* elements to facilitate the cloning of flanking genomic sequences (STELLER and PIRROTTA 1985) and the detection of gene expression patterns (O'KANE and GEHRING 1987). The known structure of the *P* element facilitates the identification of the location of new mutations and allows rapid mapping, complementation testing and cloning of affected genes. As the mutations provide links between genetic and the physical maps, they offer considerable potential for the analysis of the *Drosophila* genome.

The rich genetic background of *D. melanogaster* makes it a powerful model in the study of conserved gene function. In recognition of this potential, several groups have initiated projects to obtain a physical map of the entire euchromatic genome. This has resulted in maps comprised of ordered arrays of cloned DNA fragments in cosmids (SIDÉN-KIAMOS *et al.* 1990; KAFATOS *et al.*

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1991; MADUEÑO *et al.* 1995), YACs (GARZA *et al.* 1989), and P1 clones (SMOLLER *et al.* 1991), and the combination of this data into a reference database (reviewed by HOHEISEL *et al.* 1991; MERRIAM *et al.* 1991; HARTL and LOZOVSKAYA 1992; SPRADLING *et al.* 1995; RUBIN 1996). Two major consortia of laboratories, the Berkeley Drosophila Genome Project (BDGP) (SPRADLING *et al.* 1995; RUBIN 1996) and the European Drosophila Genome Project (EDGP) (SIDÉN-KIAMOS *et al.* 1990; KAFATOS *et al.* 1991; MADUEÑO *et al.* 1995) have the ultimate objective of determining the sequence of the euchromatic part of the genome. Inherent to the specific strategy used to build the P1 map that forms the basis of the BDGP has been the determination of "sequence tagged sites" (STSs) (OLSON *et al.* 1989) that provide short sequences at known positions along the physical map (RUBIN 1996). STS markers are also being determined by the EDGP that are themselves providing linkage with the genetic map and identifying new genes (MADUEÑO *et al.* 1995; LOUIS *et al.* 1997).

The value of the Drosophila genome projects would however, be enormously enhanced by a means of correlating genetic and physical maps to facilitate the future assignment of function to novel genes having homologues in humans and other organisms. This has been achieved to a limited extent by the fortuitous identification of known genes in the STSs and by localizing the positions of specific previously characterized Drosophila genes within cloned DNAs along the physical map (see for example, KAFATOS *et al.* 1991). However, the value of tying the two maps together by determining the insertion sites of transposable elements responsible for lethal mutations has been recognized (SPRADLING *et al.* 1996). In this article, we describe the isolation and preliminary characterization of a collection of 2460 *P*-element insertion lines on the third chromosome of *D. melanogaster*. The lethal and strong semi-lethal phenotypes of the mutations indicate that they affect genes that code for essential functions, required for either the vitality or fertility of the organism. They provide both genetic and physical markers along the length of third chromosome. In a detailed genetic and physical analysis of the region 86E-87F we demonstrate the value of such a collection in attaining the longer term objectives of the genome projects.

MATERIALS AND METHODS

Strains: Wild-type and mutant strains were maintained and mated on standard yeast-cornmeal-agar medium and all experiments were performed at 25°. All genetic markers used are described in LINDSEY and ZIMM (1992). A *y w P-lacW* stock was kindly provided by YUH NUNG JAN. A stock carrying the transposase source, *P(γ⁺Δ2-3)* on a *TM3*, *Sb* balancer chromosome and deficiency stocks *Df(3R)E229*, *Df(3R)E307* and *Df(3R)kar-Sz28* were provided by JÁNOS GAUSZ. Both lines were isogenized for the third chromosome before mutagenesis to eliminate background lethal mutations. Deficiency stocks *Df(3R)T61* and *Df(3R)I26c* were obtained from the

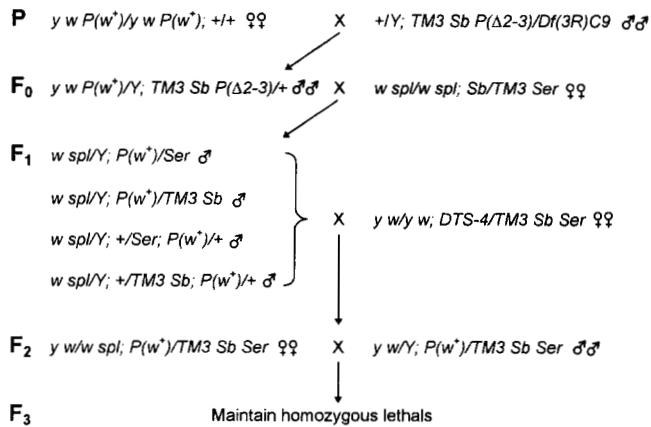


FIGURE 1.—Scheme for the isolation of third chromosome lethal *P*-element insertions. *P(w⁺)* represents the *P-lacW* transposon carrying the *w⁺* marker gene. For details see text.

Umeå Stock Center. Previously characterized mutations were also obtained from the Bloomington, Bowling Green and Umeå stock centers. A *TM6C*, *Sb Tb* stock was kindly provided by MAURIZIO GATTI. The *P*-element insertion lines are available from an outstation of the Umeå Stock Center at Szeged, Hungary (Department of Genetics, Attila Jozsef University, Kőzépfásor 52, Szeged, H-6726, Hungary; e-mail address: sidonja@biocom.bio.u-szeged.hu).

P-element mutagenesis: The genetic scheme for mobilization of *P-lacW*s shown in Figure 1. Males carrying the transposase source *P(Δ2-3)* were crossed en masse to *yellow white* females that were homozygous for a *P-lacW* insertion on the *X* chromosome. Males that carried *P-lacW* on the *X* and *Δ2-3* on the third were collected from this cross. These *F₀* "jumpstart" males were crossed in groups of 10–15 to 20–25 females of *w spl; Sb/TM3, Ser* genotype. Male *F₁* progeny with pigmented eyes indicated that the *P-lacW* has jumped to an autosome. An average of 10–15 males from each *F₀* cross were crossed individually to *y w; DTS-4/TM3, Sb Ser* females, that all third chromosomal insertions resulted in balanced *F₂* stocks. Insertions on other autosomes yielded white-eyed flies in the *F₂* generation and were eliminated. The balanced third chromosomal insertions were tested for lethality in the next generation by placing four to six pairs of *y w; P-lacW/TM3, Sb Ser* flies in a vial and examining their progeny for the presence of homozygous *P-lacW* flies. Finally, 3100 lethal or strong semi-lethal lines were identified and maintained. Each line is assigned a number representing the *F₀* cross, separated from a second number representing individual *F₁* males from a given *F₀* cross.

Cluster analysis: Since an average of 10–15 *F₁* males per *F₀* cross were used to establish individual insertion lines, some of these represented mutant clusters due to premeiotic insertions of the *P* element. To identify such clusters, complementation tests were performed among lines originated from a *F₀* cross. Usually, one line was crossed to all the other lines within such a group. About 600 clusters were identified and eliminated from the collection.

Lethal phase determination: To analyze the lethal phase, the *TM3, Sb Ser* balancer was replaced by the *TM6C, Tb Sb* chromosome. In such a genetic background, homozygous mutants can be identified by their wild-type body length, while heterozygotes have shorter body characteristic of *Tb* phenotype. An average of 10–15 pairs of flies was placed in vials supplemented with yeast paste, and eggs were collected from each line for 1 day. The development of 50–100 progeny was monitored, and the presence of homozygotes was recorded.

in all developmental stages. Lethal phase was assigned to a developmental stage in which homozygote animals last appeared. Due to uncertainties in determining the *Tb* phenotype in first larval stage, embryo and first instar lethals appear as "early lethals" in Table 1.

In situ hybridization: Females from *TM6C*, *Tb* *Sb* balanced insertion lines were crossed to wild-type (*Canton S*) males. Salivary glands of *Tb*⁺ wondering third stage larvae were dissected in 0.8% saline solution and fixed in 45% acetic acid. All treatments and hybridization procedures were performed as described earlier (SAUNDERS *et al.* 1989). Biotin-labeled DNA probe was made from the *P-lacW* plasmid (BIER *et al.* 1989) or isolated plasmid rescue sequences. After signal detection, the chromosomes were stained with Giemsa (Sigma) and mounted in DPX (BDH). The preparations were examined using phase contrast optics and signals were assigned to chromosome bands referring to LEFEVRE (1976).

Molecular techniques: Genomic DNA was isolated from 150–200 flies by the method of JOWETT (1986). Plasmid rescue from genomic DNA was performed according to PIRROTTA (1986). Other molecular techniques were performed by standard procedures as in SAMBROOK *et al.* (1989). Oligonucleotides were synthesized and used to PCR amplify sequences of the following cloned genes for use as probes to screen cosmid and P1 mini-library filters: *Taf30α*, the α - γ element, *GstD27*, *rosy*, *Ace*, and *Act87E*.

DNA sequencing: Oligonucleotides complementary to sequences close to the 3'-(5'-TCACTCGCACTTATTGCAAG CATACTG-3') and 5'-ends (5'-GCTATCGACGGGACCACCTT 3') of the *P* element were used to obtain sequences of the insertion site using an ABI PRISM 377 sequencer. DNA was sequenced using double stranded templates and dye terminator cycle sequencing as described in the Perkin Elmer ABI PRISM sequencing kit. Sequences were obtained and analyzed by the sequence analysis program supplied with the machine.

STS analysis: More than 80% of the rescued clones yielded readable sequences with an average length of 411 bp. All sequences obtained were analyzed to determine possible sequence similarities to sequences already present in nucleic acid databases. Searches were performed using the BLASTN program (ALTSCHUL *et al.* 1990) with the NCBI combined nucleic acid database.

RESULTS

Isolation of *P*-insertion mutants and lethal phase determination: To approach saturation of the third chromosome with mutations resulting from the mobilization of a *P* element, we undertook a large scale mutagenesis using the *P-lacW* transposon (see MATERIALS AND METHODS for details). The mutator element, *P-lacW*, was mobilized in our mutagenesis experiments in the presence of a stable transposase source, the *P(Δ2-3)*. Random transpositions of the mutator elements were captured in lines lacking any transposase activity. This resulted in more than 41,000 insertion lines of which approximately one-half were on chromosome 3. Originally some 3100 lethal or strong semi-lethal lines were identified. During preliminary characterization we eliminated unstable lines and members of clusters leaving 2460 lines to be characterized. These were maintained against the *TM3* and *TM6C* balancer chromosomes. We have classified these mutants according to their lethal phase as shown in Table 1 (MATERIALS AND METHODS).

TABLE 1
Lethal phase distribution in the collection

Lethal phase	No. of lines
Early lethals	1145 (46.5)
Second instar lethals	117 (4.8)
Third instar lethals	133 (5.4)
Prepupal lethals	54 (2.2)
Pupal lethals	207 (8.4)
Pharate adult lethals	312 (12.7)
Semilethals	449 (18.3)
Viables	14 (0.5)
Not determined	29 (1.2)

Values in parentheses are percentages.

The proportions of mutants showing lethality at the various stages of development are strikingly similar to those in a similar collection of 2711 lethal and semi-lethal *P*-element-generated mutations on chromosome 2 (TÖRÖK *et al.* 1993).

Cytological mapping of mutants: We have carried out two types of cytological mapping of the mutants in the collection: complementation tests against cytologically characterized chromosome deficiencies and *in situ* hybridization of *P*-element probes to salivary gland chromosomes of the mutants. A minimal set of ~60 chromosome deficiencies has been identified that together uncover some two-thirds of the euchromatin of the third chromosome. We have carried out complementation tests between the mutant lines and 28 out of this minimal set of deficiencies, representing about 34% of the euchromatin. In this way we have defined the chromosomal localization of mutations in 599 lines. In 468 lines uncovered by 21 of these deficiencies we define a total of 145 complementation groups (Table 2). Extrapolating to the whole of chromosome 3, this leads to an estimate of the total number of lethal and semi-lethal complementation groups that can be readily mutated by *P* mutagenesis in a screen of this scale as being in the order of 800.

We have applied the second mapping approach, *in situ* hybridization, to more than 1200 lines from the collection. In this randomly selected set of mutations we have found *P*-element insertions in 89% of all lettered subdivisions of the Bridges' map (Figure 2). In the majority of cases, these represent single *P*-element insertions, only 10% of lines having multiple (two or three) insertions. A compilation of these cytological data is presented in APPENDIX A.

P-insertion mutants in 86E-87F: We wished to correlate the distribution of *P*-element induced mutants with the genetic map of a well characterized region of the third chromosome. We therefore chose to undertake a detailed analysis of the region 86E-87F since it has been subject to saturation chemical and X-ray mutagenesis in the studies of CHOVNICK *et al.* (1976), HILLIKER *et al.* (1980) and GAUSZ *et al.* (1981). The breakpoints of

TABLE 2

Groups of noncomplementing mutations within the collection uncovered by chromosomal deficiencies

Deficiency	Breakpoints	No. of bands uncovered	No. of noncomplementing lines	No. of complementation groups
Df(3L)HR119	63C6;63E9	9	7	ND
Df(3L)GN50	63E1-2;64B17	44	14	9
Df(3L)pbl-XI	65F3;66B10	40	4	3
Df(3L)ZP-1	66A17-20;66C1-5	18	10	5
Df(3L)66C-G28	66B8-9;66C9-10	14	8	3
Df(3L)h-i22	66D10-11;66E1-2	6	11	4
Df(3L)29A6	66F5;67B1	13	8	2
Df(3L)AC1	67A2;67D13	43	6	3
Df(3L)vin7	68C8-11;69B4-5	32	29	8
Df(3L)fz-GF3b	70C1-2;70D4-5	18	14	9
Df(3L)fz-D21	70D3;70E3-8	9	13	ND
Df(3L)Brd12	70E;71B	15	28	ND
Df(3L)st-f13	72C1-2;73A3-4	26	14	ND
Df(3L)81k19	73A3;74A	60	24	12
Df(3L)W10	75A6-7;75C1-2	17	22	4
Df(3L)Cat	75C1-2;75F1	31	51	7
Df(3L)ri-79c	77B1-C7;77F1-78A7	22	14	8
Df(3R)Antp17	84B1-2;84D11-12	24	20	ND
Df(3R)by62	85D11-14;85F16	45	93	14
Df(3R)T61	86E3-4;87A7-9	31	34	10
Df(3R)E229	86F6-7;87B1-2	16	4	4
Df(3R)E307	87B2-4;87C9-D3	20	20	11
Df(3R)kar-Sz28	87C7-8;87E9-10	25	17	8
Df(3R)126c	87D14-E1;87F11-12	23	18	10
Df(3R)sbd26	89B9-10;89C7-D1	22	30	ND
Df(3R)P14	90C2-D1;91A17-18	50	40	ND
Df(3R)crb87-5	95F7-9;96A17-18	24	23	8
Df(3R)A117der21	100A3-7;100B8-9	13	23	8

ND, not determined.

many deficiencies have been mapped within this interval, permitting the detailed positioning of a large number of the previously obtained lethal mutations. We were able to obtain representative alleles of 62 of these complementation groups. We chose to localize *P*-element-induced mutations from the collection with respect to five overlapping deficiencies that delineate the region. The results of this complementation analysis are shown in Figure 3. A total of 87 lines were identified within the collection that fell into 38 complementation groups within the interval. The cytogenetic mapping of 21 of these complementation groups was consistent with the localization of the *P*-element insertion(s) by *in situ* hybridization. Of these, we were able to identify eight complementation groups that corresponded to previously identified genes within the set of 62 that we tested (Figure 4). *P*-element-induced mutations in 17 complementation groups appeared not to have associated *P* elements that could be identified by *in situ* hybridization (Figure 3D). In all of these cases strong hybridization of a *P*-element probe could be seen at another chromosomal site. We identified nine apparently novel complementation groups within this group of 17 genes. We noted that in those cases that appeared

to be "hot-spots" for mutagenesis by the large number of *P*-induced alleles that had been generated, only a small proportion of the mutants had *P* elements at the site of the gene. The *prospero* locus, for example, is represented by 14 *P*-induced alleles, of which only two have *P* elements at the locus.

Conversely, in the studies to localize *P* elements within 1200 randomly selected lines by *in situ* hybridization as described above, we were able to identify 40 independent insertions within the 86E-87F interval that did not correspond to lethal complementation groups mapping to this region. These lines carry lethal or semi-lethal mutations elsewhere on the chromosome.

Correlation of the *P*-insertion sites with the molecular genetic map of the 86E-87F interval: The *E. coli* replication origin and selectable marker present in *P-lacW* facilitates the isolation of *P*-element sequences linked to flanking chromosomal DNA (STELLER and PIRROTTA 1985; PIRROTTA 1986). We have utilized chromosomal sequences isolated in this way in direct hybridization experiments to localize the sites of *P*-element insertions on the molecular maps from the EDGP and BDGP. We used a divisional set of cosmids from the EDGP that comprises 261 clones previously selected from a total

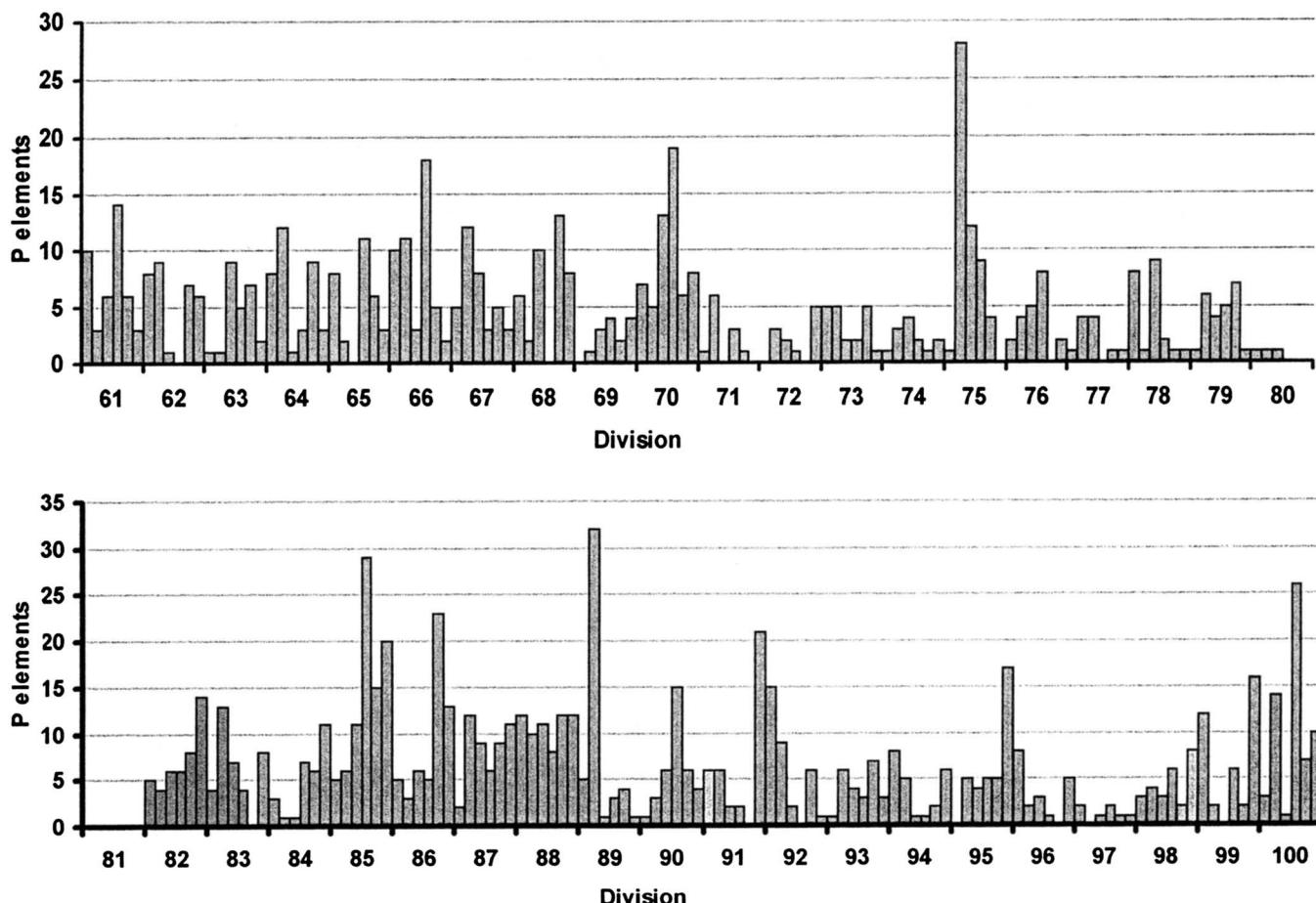


FIGURE 2.—Localization of *P*-element insertions in 1200 mutant lines from the collection. The histograms display numbers of lines in which *P*-element probes hybridize *in situ* to the six lettered subdivisions in the indicated numerical divisions of the Bridges' salivary gland chromosome map for the left (upper panel) and right (lower panel) arms of chromosome 3 (BRIDGES 1935, 1941). A full representation of this data is provided in APPENDIX A.

genome library by their ability to hybridize with a microdissected region specific probe (SAUNDERS *et al.* 1989). At the outset of our work, cosmids from this set had been ordered into a series of 44 contigs by restriction enzyme fingerprinting followed by computer analysis using the approaches of COULSON and colleagues (1986). One hundred fifteen cosmids remained unattached to contigs. We made replica filters carrying this set of cosmids, and 34 P1 clones that were a subset of 173 P1 clones identified by the BDGP as carrying DNA from this region, linked by virtue of sharing STS elements. We selected these 34 P1 clones as representing a minimal set that would cover the interval according to the BDGP map. Hybridization probes were prepared from DNA flanking *P* insertions rescued from 35 mutant lines, of which 15 carried lethal complementation groups within the interval. In each case, we confirmed that the rescued DNA hybridized *in situ* to the correct chromosomal position (APPENDIX B). As additional hybridization probes we obtained 16 previously cloned genes that had been mapped to the region. We then performed sequential colony hybridizations to a number of filter replicas (GRUNSTEIN and HOGNESS 1975).

The results, given in detail in APPENDIX B, are summarized in Figure 5. We found the density of *P*-element insertions to be such that the flanking sequences frequently hybridized to a number of cosmid contigs. This enabled us to combine the contigs into seven super-contigs that cover most of the interval. One of these super-contigs is comprised of 18 of the original cosmid contigs linked at one site through a P1 clone. This super-contig appears to extend from position 86E4-8 to 87C1-3, an estimated distance of 800 kb from the maps of HEINO *et al.* (1994).

STSs at *P*-element insertion sites in 86E-87F: We have determined STSs corresponding to the chromosomal sites immediately flanking one side of the *P* element in each of 49 lines with insertions in this interval. As *P* elements have a strong tendency to insert into noncoding sequences to the 5' of genes and at a lower frequency into the coding region itself, it seemed that some of these STSs might mark or extend into coding sequence in at most 50% of the rescued DNA. In seven cases we detected sequences of mainly the *P*-element vector itself, and conclude either that the segment of rescued DNA contained only an extremely short seg-

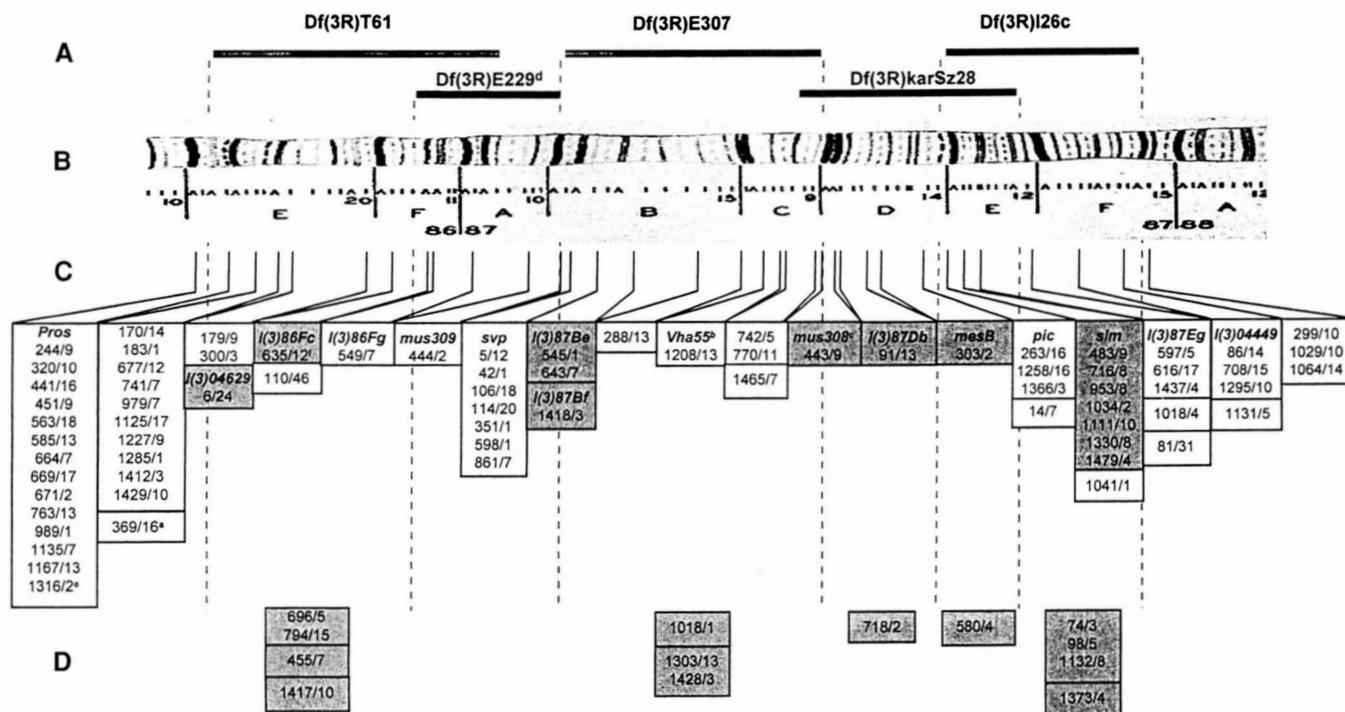


FIGURE 3.—Lethal complementation groups with *P*-element-induced alleles lying within 86E-87F. (A) The minimal set of chromosome deficiencies that uncover the interval. (B) Cytology of the region taken from BRIDGES (1935, 1941). (C) Complementation groups (boxed) for which one or more alleles show *P* elements at the corresponding cytological position by *in situ* hybridization. These are connected by solid lines to their cytological positions determined by *in situ* hybridization with a *P*-element probe. Shaded boxes represent genes for which there was no detectable *P*-element in the region by *in situ* hybridization, but which had previously been mapped with respect to deficiency breakpoints by other laboratories (data from FLYBASE). (D) Remaining complementation groups for which *P* elements could not be detected within the region by *in situ* hybridization. These correspond to novel genes that we have mapped to complementation groups within the broad intervals defined by the minimal set of chromosomal deficiencies. ^aThe mutation in line 369/16 was uncovered by *Df(3R)E229*, but the *P*-element hybridizes to 86E1–2. ^bThe mutation in line 1208/13 is not complemented by *I(3)87Bi* and yet the *P*-element hybridizes *in situ* to 87C1,3. It also fails to complement a mutation in the *Vha55* gene (formerly named *I(3)87Ca*). An STS from the rescued flanking sequences shows identity to the *Vha55* coding sequence (Table 3). ^cAll previously described alleles of *mus308* are viable (HARRIS *et al.* 1996) and yet this mutant fails to complement a line of *mus308* obtained from the Bloomington stock center. ^dThe distal breakpoint of *Df(3R)E229* and the proximal breakpoint of *Df(3R)E307* are cytologically indistinguishable. However, they are separated by a small genetic interval that includes one lethal complementation group (JANOS GAUSZ, personal communication). ^eLine 1316/2 carries a second site mutation in *I(3)87Ab*. ^fLine 635/12 carries a second site mutation in *I(3)86Ff*.

ment of flanking chromosomal sequence, or that there were multiple *P* elements at the insertion site. In 28 cases, BLAST searches revealed no significant homologies with known genes. In four cases, the STS element corresponds to an STS determined by BDGP, and in 10 lines we detect significant homologies with known genes or genetic elements that offer yet further linkage between the genetic and physical maps (Table 3). Of these 10 matches, three correspond to either repetitive sequences or mobile elements, and seven correspond to putative single copy protein coding sequences. Mutations have previously been described in the *Drosophila* genes for five of these proteins. However two correspond to *Drosophila* genes identified only by homology to genes from other organisms and for which mutations are not available. These genes encode a cytochrome c oxidase subunit and a member of the myoD family of transcriptional regulators. It will be of interest to determine whether indeed the inserted *P* elements affect the expression of these two predicted proteins.

DISCUSSION

It is our hope that this collection of *P* insertions on chromosome 3 of *D. melanogaster* will provide a valuable resource for the fly community. It provides an asset that can be screened directly for novel mutants in which particular aspects of cell biology or development are affected. The accompanying article exemplifies the value of the collection in a screen of this type for mutations affecting the embryonic development of the peripheral nervous system (SALZBERG *et al.* 1997). This study identified 96 mutations corresponding to 19 known genes that had mainly been identified through chemical mutagenesis, together with seven novel complementation groups. We have also screened the collection for mutations affecting mitosis and male meiosis. We have identified some 44 complementation groups in which mutants display defective mitosis in larval neuroblasts, and an equivalent number affecting meiosis in males (in preparation). Moreover we have identified 20

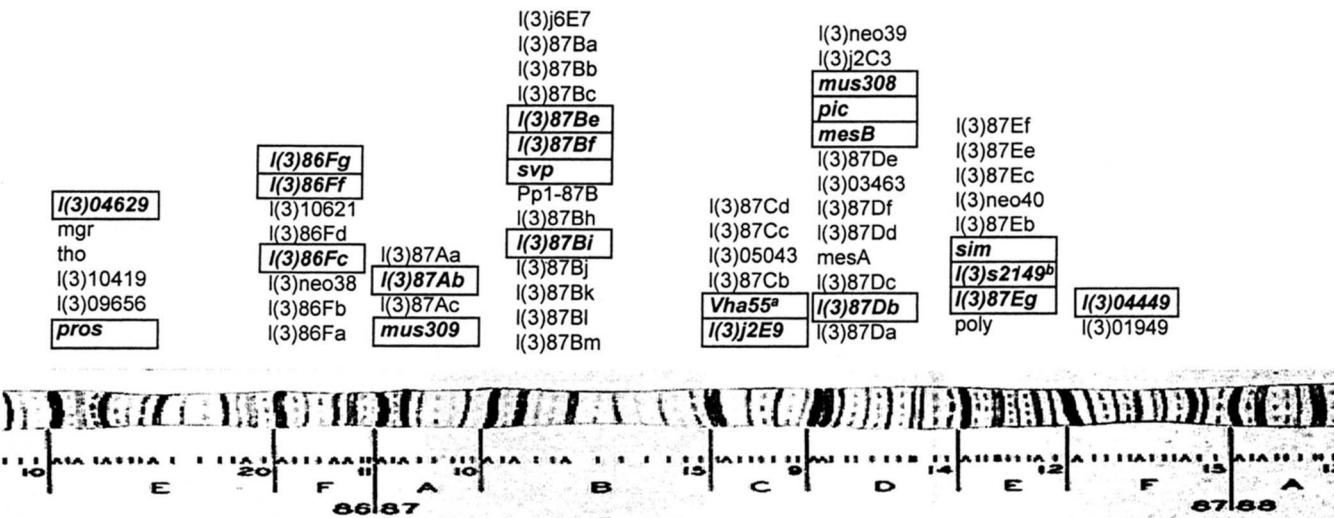


FIGURE 4.—Previously identified mutants used for complementation tests with *P*-element-derived mutants within the 86E-87F interval. The positions of the genes are indicated only with respect to the lettered subdivisions of the cytological map (data taken from FLYBASE). Loci for which a *P*-element-induced allele was detected from the collection are boxed. ^aLine 1208/13 fails to complement both *I(3)j2E9* and *I(3)87Ca*. These are therefore alleles of *Vha55*. ^bLine 616/17 fails to complement both *I(3)s2149* and *I(3)87Eg*. These are therefore allelic.

or so female sterile mutations in the collection that when homozygous produce embryos showing mitotic defects (in preparation). The collection may also be easily screened for mutations that act as modifiers of dominant mutations that result in developmental defects. In this way the collection has been used for the identification of the first mutant alleles of a gene, *daughter of sevenless* (*dos*), that act as suppressors of the activated SEV tyrosine kinase receptor in eye development (RAABE *et al.* 1996). The localization of *P* elements in 89% of lettered subdivisions identifies entry points for further genetic dissection along the whole of the chromosome. Specific strains from the collection having *P* elements mapped to a particular site may be tested by complementation to determine whether they are alleles of known genes in such a region. If an allele is not found, it may be possible to generate one by *P*-element mobilization and selection of new mutations in the vicinity of the original element, so-called “local hopping” (TOWER *et al.* 1993; ZHANG and SPRADLING 1993). Alternatively male-recombination in a dysgenic background can be used to generate deletions extending from the *P*-element insertion site and so help in the molecular delineation of the region (PRESTON *et al.* 1996; PRESTON and ENGELS 1996). The collection may also be used for reverse genetics in cases where a cloned *Drosophila* gene has not yet been correlated with a mutant phenotype. An effective approach to this end involves the use of PCR between primers from a sequenced gene and the *P*-element to provide a molecular link between that gene and a mutagenizing transposon (BALLINGER and BENZER 1989; KAISER and GOODWIN 1990). This strategy was recently adapted for use on a similar collection of *P*-element insertion mutants on the second chromosome (GUO *et al.* 1997).

One advantage of having a *P*-element-induced allele of a particular gene is the ease whereby it permits the molecular cloning of that gene. However, the incidence with which we find second site mutations where *P* elements may no longer be associated with lethality necessitates some precautionary measures before molecular studies are undertaken using these lines. It is important to carry out both *in situ* hybridization studies and cytogenetic mapping against a chromosomal deficiency, preferably in association with reversion tests to determine whether under dysgenic conditions, the *w⁺* phenotype associated with the *P* element will revert together with the lethal mutation carried on the chromosome. Nevertheless ~50% of lethal and semi-lethal complementation groups appear to have alleles that retain *P* elements that should provide ready molecular access to the corresponding gene. Our mutagenesis has resulted in the generation of new alleles in 21 out of 62 known genes in the cytological interval 86E-87F. In total the screen has generated mutations in 38 complementation groups from this interval. *P*-elements appear to be associated with 21 out of these 38 genes by the criterion of *in situ* hybridization of a *P*-element probe to the cytogenetic interval to which the mutation is mapped. In 18 of these 21 cases there seems to be only a single *P*-element insertion on the chromosome. On the other hand, we identify some 17 complementation groups for which lethality in 86E-87F is no longer associated with a *P* element in the region, as well as 40 examples of *P* insertions in the interval that are linked to mutations elsewhere on the chromosome. We consider two main possibilities for the genesis of such mutations. First that they occur following the insertion of a *P* element into a hotspot followed by its rapid excision and reinsertion at a second site, leaving a mutation at the

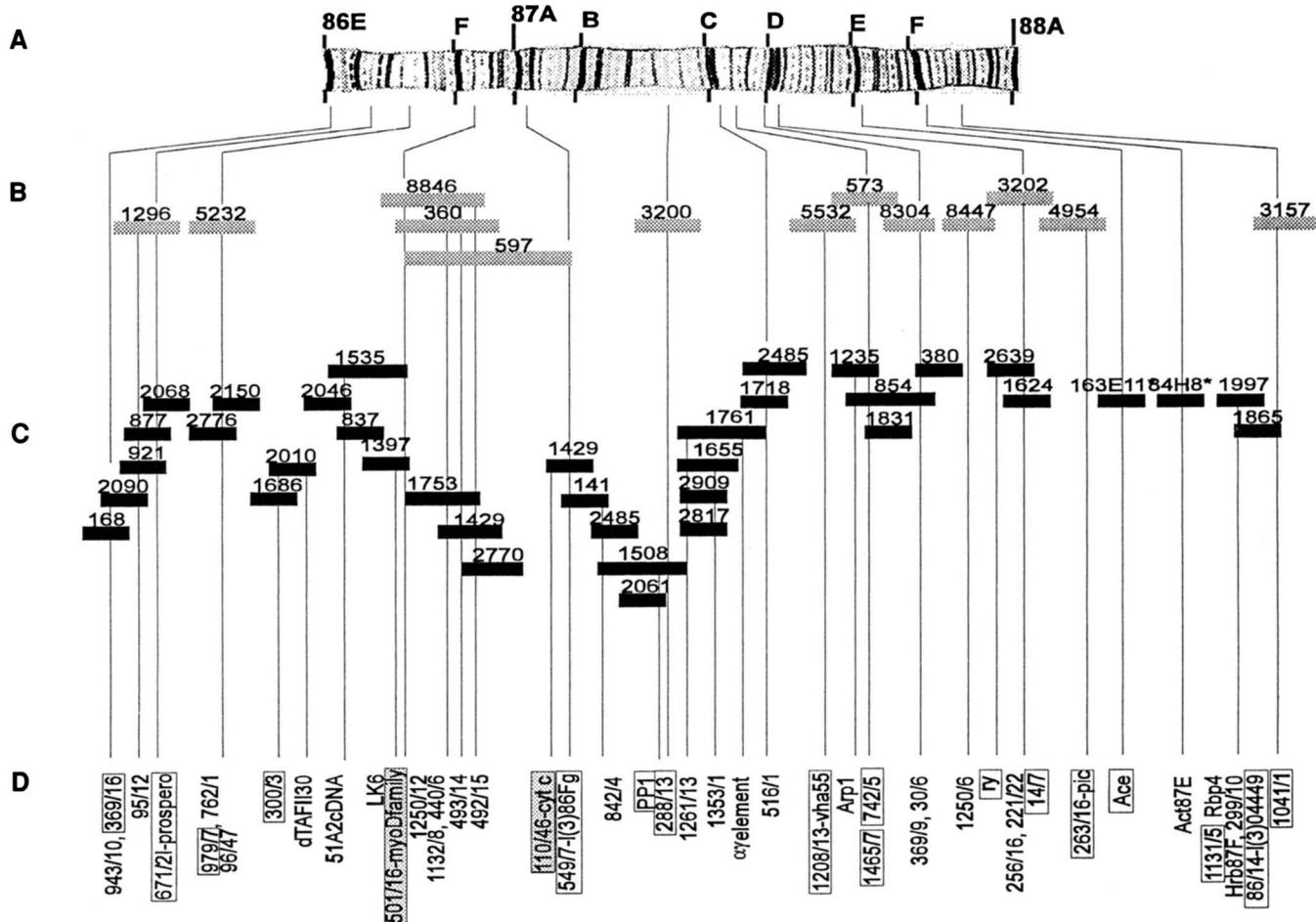


FIGURE 5.—Linking cytogenetic and physical maps in the interval 86E-87F. (A) Cytology of the region taken from BRIDGES (1935, 1941). The *in situ* hybridization sites are shown for several rescued chromosomal DNAs flanking *P* insertions and several cloned genes. A complete set of data may be found in APPENDIX B. The chromosomal *in situ* hybridization site is tied by the “dog-legged” vertical lines that pass through hybridizing P1 clones and cosmid contigs to the hybridization probe in row D. (B) Individual P1 clones from the BDGP that show hybridization to the probes used in this experiment (see text for details). (C) Cosmid contigs from the EDGP in which one or more individual cosmids show hybridization. Details of individual cosmids together with the identification of unattached cosmids that show hybridization are given in APPENDIX B. In most cases these data permit several unattached cosmids to be brought into contigs. Two exceptional individual cosmids that remained unattached are indicated on this figure by asterisks. (D) Rescued *P* elements and flanking chromosomal DNA and cloned genes used as hybridization probes in this experiment. Those corresponding to a genetic complementation group are boxed. Shaded boxes represent mutants of new genes identified solely by homology of their coding sequence to genes from other organisms. All hybridization linkages between probes, cosmid contigs, and individual P1 clones are shown by the vertical lines. Lengths of horizontal lines indicating P1 clones and cosmid contigs are not drawn in proportion to physical distance.

site of original insertion and excision. The generation of 14 *prospero* alleles of which only two have *P* insertions would be consistent with such a hypothesis. A second possibility is that the P-M dysgenic cross also mobilized transposons other than *P* elements that result in mutations not associated with *P* insertions carried on the same chromosome as the *P*-insertion events that have been selected.

The hybridization of chromosomal sequences flanking *P*-element insertions in the 86E-87F interval to recombinant cosmids and P1 clones from the EDGP and BDGP, respectively, not only provides linkage between the two molecular maps, but also effectively ties together the genetic and physical maps within this cyto-

logical interval. The correspondence between the physical map positions of chromosomal sequences flanking lethal *P*-element insertions and the cytogenetic locations of the mutations is excellent. These hybridization experiments were extremely effective at linking cosmid contigs, thus reducing the number of contigs from 44 at the outset to seven following this analysis. The large number of original contigs reflects the method used to establish contigs; a computer-generated comparison of restriction fragment fingerprints. This approach is only able to recognize overlaps between clones where the overlaps are large enough to generate sufficient identical restriction fragments. Direct nucleic acid hybridization, on the other hand, can detect much smaller over-

TABLE 3
Known genes and sequences identified by STSs

STS	Length	Homologous sequences	High score	Smallest sum probability
5/12	872	<i>D. melanogaster seven-up</i>	1494	2.1e-174
109/17	149	<i>D. melanogaster</i> P1 clone DS04219	363	1.4e-30
110/46	727	<i>D. melanogaster</i> cytochrome C oxidase subunit	387	3.0e-63
501/16	391	<i>R. norvegicus</i> myogenic regulatory factor	204	9.1e-07
516/1	586	<i>D. melanogaster</i> α - γ element	425	7.6e-30
616/17	722	<i>D. melanogaster</i> P1 clone	1218	2.8e-162
671/2	213	<i>D. melanogaster</i> <i>prospero</i>	166	3.2e-15
1019/2	159	<i>D. melanogaster</i> P1 clone DS04219	313	1.0e-26
1084/8	281	<i>D. melanogaster</i> <i>abnormal wing disc</i>	572	2.8e-54
1208/13	264	<i>D. melanogaster</i> vacuolar ATPase B subunit	429	2.2e-26
1250/6	418	<i>D. melanogaster</i> mobile element BS	301	5.5e-22
1263/13	313	<i>D. melanogaster</i> <i>buttonless</i>	859	9.9e-64
1303/13	398	<i>D. melanogaster</i> P1 clone DS04219	1002	1.7e-130
1353/1	259	<i>D. melanogaster</i> mobile element <i>hoppel</i>	257	8.3e-21

The following STSs did not show significant homology to database entries: 30/6, 53/2, 58/18, 81/31, 95/12, 96/47, 110/41, 156/16, 263/16, 288/13, 299/10, 300/3, 369/16, 492/15, 549/7, 742/5, 805/3, 842/4, 943/10, 979/7, 1018/4, 1107/9, 1131/5, 1442/7, and 1465/7. STSs of 86/14, 221/22, 483/10, 762/1 and 1083/10 proved to be part of the *P*-element vector.

laps. The complementarity of the approaches of the two major genome mapping projects is also illustrated by these data; there are examples both of hybridization probes that would detect only P1 clones, and probes that would detect only cosmid clones. The linkage of the two physical maps will offer distinct advantages for the complete DNA sequencing of this chromosomal division.

Linkage between the genetic and physical maps was also provided by the analysis of STSs from the sites of *P*-element insertions. As STSs were only determined from one side of insertions, we could expect the possibility of the STS hitting a recognizable protein coding sequence in only half of these cases. Moreover, *P* elements most often insert in the transcriptional regulatory sequences to the 5' of the gene from which position it would seem unlikely that the STS might extend into coding sequence. However, *P* elements are also known to insert into coding regions, and consequently we were able to detect homology to seven recognizable proteins within the 42 STSs representing chromosomal DNA sequence. Mutations have already been described in five of these genes, but the *P*-element-induced mutants could offer real possibilities for studying the function of the other two genes within the whole organism. These encode fly homologues of a cytochrome c oxidase subunit and a member of the myoD family of transcription factors, identified only through sequence homology. However, we note there are 16 further lethal complementation groups from the 86E-87F region within the collection that are either novel or for which the gene has not been cloned, where *P* elements are present at or close to the locus. These insertion sites represent excellent candidates for more extensive sequencing of

chromosomal DNA that should uncover coding sequences whose functions may be studied through characterization of the corresponding mutant phenotypes.

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APPENDIX A

Line	Lethal phase	Location	Deficiency	Line	Lethal phase	Location	Deficiency
0111/30	E-L1	61A		0843/07	L3	62B4-5	
0960/03	P	61A		0186/11	E	62B4-9	<i>Df(3L)GN50</i>
1040/11	E	61A		1146/02	E	62B9-12	
1460/09	E	61A	<i>Df(3R)A117der21</i>	0020/01	pP-P-pA	62C	
1418/03	P	61A	<i>Df(3R)E307</i>	0725/17	P-pA	62E	
0741/07	P	61A	<i>Df(3R)T61</i>	0872/17	P	62E	
0833/11	L3	61A and 61A and 86E		1019/04	A	62E	
0492/17	A	61A, 63C and 76C	<i>Df(3R)by62</i>	1130/03	E	62E	<i>Df(3R)A117der21</i>
1042/07	E	61A1-3		0281/13	P	62E and 64F	
1444/05	A	61A1-3, 66B and 70D		0688/02	A	62E4-9	
0751/02	Viable	61B		0651/09	pA	62E6-9	
1319/09	P-pA-A	61B		0828/08	E	62F	
0702/09	pA-A	61B1-3		1037/07	L3	62F	
0574/01	A	61C		0939/09	A	62F	
0652/08	A	61C1-4 and 85D7- 13		1122/02		62F	<i>Df(3L)ri-79c</i>
1422/15	pA	61C3-5		1040/18	E	62F and 99F	
0419/19	A	61C5-9		0852/12	L2	62F1-2	
0965/07	L3	61C5-9		0917/09	E	63A1-3 and 63E1-6	
0953/08	E	61C5-9	<i>Df(3R)kar-Sz28</i>	1030/12	E	63B	<i>Df(3L)ZP-1</i>
0587/01	E	61D		1477/03	P-pA	63C	
1077/05	A	61D		0424/15	P-pA	63C	
0094/26	E	61D		0225/58	A	63C	
0098/26	A	61D		0989/05	pP-P	63C	
0203/10	E	61D		1283/06	A	63C	
0580/04	A	61D	<i>Df(3R)126C and</i> <i>Df(3R)kar-Sz28</i>	0787/09	pA-A	63C and 73D	
0794/15	E	61D	<i>Df(3R)T61</i>	1106/10	A	63C and 85F1-3	
1438/14	pA	61D and 69F		0364/01	A	63C4-6 and 85D	<i>Df(3L)ZP-1</i>
0730/06	pA-A	61D and 83A		1296/15	A	63D	
1004/03	A	61D1-2		1037/10	E	63D	
1483/07	A	61D1-2		0671/11	A	63D	
0307/04	E-L1	61D1-2		0439/25	E	63D	
1469/12	A	61D1-3		0473/24	P-pA	63D and 80B	
0368/10	A	61E		0422/07	A	63E	
0040/19	A	61E		0494/13	E	63E	<i>Df(3L)cat</i>
0074/12	pA	61E		1328/02	E	63E	<i>Df(3L)vin7</i>
1372/03	A	61E		1322/09	E	63E	<i>Df(3R)A117der21</i>
0264/38	E	61E and 65A		0004/17	E	63E and 83B	<i>Df(3R)A117der21</i>
0264/37	A	61E1-2		0510/22	E	63E1-2; 64B17	
1386/06	P	61F1-2		0638/13	E	63F3-7	
0036/06	pA	61F5-8		0639/13	E	63F3-7	
0959/14	E	61F8		0879/02	A	64A	
1041/03	L3-pP	62A		1224/04	E	64A	<i>Df(3L)GN50</i>
0787/08	L3	62A		0143/10	L1	64A	<i>Df(3L)HR119</i>
0416/08	P	62A and 99A		1429/16		64A1-3	
1346/12	L3-pP-P	62A1-2		0510/01	E	64A1-5	<i>Df(3L)GN50</i>
0906/09	E	62A1-4		1262/15		64A1-5	<i>Df(3L)GN50</i>
1291/14	A	62A3-6		1372/12		64A4-6	<i>Df(3L)GN50</i>
1196/07	pA-A	62A4-10 and 84E	<i>Df(3L)cat</i>	1449/12		64A6-12	<i>Df(3L)GN50</i>
0422/39	A	62A6-12		1036/16	E	64B	
0473/31	A	62B		0973/08	3	64B	<i>Df(3L)GN50</i>
0041/08	E-L1	62B	<i>Df(3L)fz-FG3b</i>	0934/13	E	64B	<i>Df(3L)GN50</i>
0625/16	E	62B	<i>Df(3L)h-i22</i>	0925/01	E	64B	<i>Df(3L)GN50</i>
1010/03	E	62B	<i>Df(3L)ri-79c</i>	1483/16	A	64B1-2	
1258/01	A	62B10-12, 91F10-13		0607/06	A	64B1-2	
				1110/01	A	64B1-2	
				0407/08	L3-pP	64B1-2	<i>Df(3L)Brd-12</i>
				1400/09	E	64B1-2 and 66E	<i>Df(3L)GN50</i>
				0241/20	P-pA	64B1-6	

APPENDIX A

Continued

Line	Lethal phase	Location	Deficiency	Line	Lethal phase	Location	Deficiency
0483/09	E	64B9-15	<i>Df(3R)kar-Sz28</i>	0293/09	P	66B	<i>Df(3L)pb1-XI</i>
1364/14	pA-A	64C12-15		0230/14	L2	66B	<i>Df(3R)by62</i>
0629/14	A	64D		0649/12	A	66B1-6	
0999/08	E	64D and 88F		0459/02	pA	66B1-6	
0793/11	P	64D and 89B and 100B	<i>Df(3R)sbd26</i>	0023/09	A	66B10-13	<i>Df(3L)ZP-1</i>
0126/13	E	64E	<i>Df(3L)W10</i>	0076/08	Viable	66B3-9	<i>Df(3L)ZP-1</i>
1433/01	E	64E	<i>Df(3R)A117der21</i>	0903/14	Viable	66B3-9	<i>Df(3L)ZP-1</i>
0726/13	A	64E and 70D1-2	<i>Df(3L)fz-GF3b</i>	0989/11	pA	66B6-10	
0050/05	pA-A	64E and 70F		0088/31	pP-P	66C	
0666/19	pP-P	64E1-5		0906/13	E	66C	<i>Df(3L)Brd-12</i>
1456/09	A	64E3-10		0737/04	P-Pa	66C	<i>Df(3R)crbS87-5</i>
0964/04	pA	64E-13		0900/01	E	66D	
1335/01	P	64E4-10 and 82E1- 4		1261/07	A	66D	
1455/11	A	64E4-12		0187/17	L3	66D	
0952/03	P-pA	64F		0256/37	E	66D	<i>Df(3L)h-i22</i>
1250/15	L3	64F and 100D3-4		1483/18	E	66D	<i>Df(3L)h-i22</i>
0151/10	L1	65A		0106/05	E-L1	66D	<i>Df(3L)h-i22</i>
1038/07	E	65A		0092/23	E	66D	<i>Df(3L)h-i22</i>
1203/07	A	65A1-2		0084/18	E	66D	<i>Df(3L)h-i22</i>
0545/01	E	65A1-6	<i>Df(3R)E307</i>	1398/10	E	66D	<i>Df(3L)h-i22</i>
0718/06	A	65A10-15 and 95B		0523/18	E	66D	<i>Df(3R)P14</i>
0666/10	pP-P	65A7-12		1193/13	E	66D and 88B	<i>Df(3L)h-i22</i>
0528/13	pP-P	65A8-12		0240/20	Viable	66D and 93B	
0341/07	A	65B		0212/04	L3	66D1-2	
1429/09	P	65B		1383/07	A	66D10-15	
1143/02	pA-A	65D		1100/13	L2	66D10-15	
1043/15	E	65D		0702/04	pA-A	66D10-15	
0379/13	pA-A	65D		0635/12	E	66D10-5	<i>Df(3R)T61</i>
0939/02	A	65D		0042/09	E	66D23-27	<i>Df(3L)ZP-1</i>
0673/09	L1	65D		0369/01	A	66E	
0283/04	pA-A	65D3-6		1017/15	P-pA	66E	
0252/66	A	65D3-6		0718/02	E	66E	<i>Df(3R)kar-Sz28</i>
0759/02	A	65D4-6	<i>Df(3R)by62</i>	0327/08	E-L1	66E, 83C1-3, 96D	<i>Df(3R)by62</i>
0471/17	A	65D4-6 and 68C10- 15		1002/10	E	66F	<i>Df(3R)E307</i>
1441/14	E	65E		1018/01	P	66F	
1465/08	A	65E		0841/01	E	67A	
1422/06	A	65E1-5		0369/12	E	67A	<i>Df(3L)29A6</i>
0496/04	P-pA	65E4-7		0274/03	A	67A and 82E	
0613/18	pA-A	65E5-9, 78C, 79E1- 3		0495/08	A	67A5-9	
0121/24	A	65F		0369/16	pA	67A5-0 and 86E10- 13	
0244/21	A	65F and 77C		0621/18	P	67B	
0422/38	A	65F3-6		1004/13	E	67B	<i>Df(3L)29A6</i>
0110/27	P	66A	<i>Df(3L)pb1-XI</i>	0250/07	E	67B	<i>Df(3L)29A6</i>
0083/20	E	66A	<i>Df(3L)pb1-XI</i>	0999/05	E	67B	<i>Df(3L)29A6</i>
0726/11	E	66A	<i>Df(3L)pb1-XI</i>	0423/14	pA	67B1-10	
0542/03	P	66A	<i>Df(3R)P14</i>	0604/04	A	67B1-3	<i>Df(3L)st-f13</i>
1479/15	E	66A, 82D and 85E1-7	<i>Df(3R)kar-Sz28</i>	0604/02	A	67B1-5	
0287/05	pA-A	66A1-2		0427/05	A	67B1-5	
0669/05	A	66A1-5		0876/07	A	67B11-13	
0714/11	A	66A10-15		1351/11	pA	67B8-13	
0845/07	L3	66Ab		0446/31	A	67B9-13	
1232/06	A	66B		0893/02	E	67C	
				0952/09	A	67C1-2	
				0420/16	E	67C1-2	<i>Df(3R)A117der21</i>
				0443/39	A	67C1-4	
				0890/04	P	67C3-5	

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Line	Lethal phase	Location	Deficiency	Line	Lethal phase	Location	Deficiency
0273/42	A	67C3-6		1067/13	pA	69C4-10	
0890/13	A	67C4		0974/16	E	69D	
1115/05	L3	67C9-11		0059/12	A	69D	
0560/03	Viable	67D	<i>Df(3L)cat</i>	1068/10	P-pA	69D1	
0101/02	A	67D and 100F	<i>Df(3L)AC1</i>	0974/06	L1	69D1-3	<i>Df(3L)Brd-12</i>
1005/13	pA-A	67D8-13		0423/09	Viable	69E	
0188/12	P-pA	67E		0426/06	A	69E	
1465/07	P	67E and 87C	<i>Df(3R)kar-Sz28</i>	1479/10	P	69F3-7	
0470/03	L3	67E and 98E		1479/12	A	69F3-7 and 84D4-8	
0453/11	A	67E5-7		0916/03	E	69F4-7	
0059/16	pA	57E5-7	<i>Df(3R)P14</i>	0816/10	P-pA	70A	
0583/02	pA	67F		0428/05	A	70A	
0840/15	P	67F		0641/17	E	70A	
0896/05	L3	67F and 98F3-11		1309/01	L3	70A	
0303/05	L3	68A	<i>Df(3L)vin2</i>	0024/05	A	70A and 94B	
1253/10	pA-A	68A1-2		0473/22	pA	70A1-5	
0643/07	E	68A3-6	<i>Df(3R)E307</i>	1343/03	pA-A	70A1-5	
0069/30	A	68A3-9		1311/03	A	70B	
0426/10	A	68A4-6		0270/22	A	70B	
0871/06	A	68A4-9		0717/05	pA-A	70B and 85F10-15	
1083/06	E	68B		0492/15	A	70B and 86F and	
1135/07	E	68B and 78C1-3 and 82F	<i>Df(3R)T61</i>			97A	
0840/14	P	68C		0965/15	E	70C	
0428/06	P	68C	<i>Df(3L)cat</i>	1342/17	E	70C	<i>Df(3L)fz-GF3b</i>
1034/14	P	68C and 91F1-6 and 2L		0041/12	E-L1	70C	<i>Df(3L)fz-GF3b</i>
0487/18	A	68C1-5		0318/20	E	70C	<i>Df(3L)fz-GF3b</i>
0245/35	pA-A	68C1-5 and 88A		0670/06	pA	70C	<i>Df(3L)fz-GF3b</i>
1100/06	E	68C5-8	<i>Df(3R)sbd26</i>	1485/13	E	70C	<i>Df(3L)fz-GF3b</i>
0276/07	A	68C8-11		0277/14	L1	70C	<i>Df(3L)fz-GF3b</i>
0590/11	A	68C8-11		0284/01	pP-P	70C and 96A17-22	
1329/16	L3	68E		1366/03	L2	70C1-10	<i>Df(3R)kar-Sz28</i>
0422/28	P-pA	68E		0276/14	L3	70C1-3	<i>Df(3L)cat</i>
0040/24	A	68E		0920/12	pA-A	70C1-5	
0104/07	E	68E	<i>Df(3L)vin7</i>	0958/14	E	70C10-15	
1449/05	E	68E	<i>Df(3L)vin7</i>	0608/07	L3	70C4-8 and 94F	
0545/13	L2	68E	<i>Df(3L)vin7</i>	1295/15	A	70D	
0046/23	A	68E	<i>Df(3L)vin7</i>	1488/01	L1	70D	
0245/34	E	68E	<i>Df(3L)vin7</i>	0871/13	E	70D	
0521/06	E	68E	<i>Df(3L)vin7</i>	0238/44	A	70D	
0810/03	E	68E	<i>Df(3L)vin7</i>	0453/05	A	70D	
0475/22	E	68E	<i>Df(3L)vin7</i>	1050/13	E	70D	
0033/02	E	68E	<i>Df(3R)P14</i>	0845/12	pP	70D	
0528/04	E	68E4	<i>Df(3L)vin7</i>	0238/37	A	70D	<i>Df(3L)cat</i>
0821/06	P-pA	68F		0844/01	E	70D1-2	<i>Df(3L)fz-GF3b</i>
1357/10	P-pA-A	68F		1348/02	L3	70D1-2	<i>Df(3L)fz-GF3b</i>
1002/09	L3	68F		0848/07	A	70D1-2	<i>Df(3L)fz-GF3b</i>
1266/15	E	68F	<i>Df(3L)HR119</i>	0442/21	A	70D3-8	
1459/11	E	68F	<i>Df(3L)vin7</i>	0920/06	P	70D4-5	
0073/29	E-P	68F	<i>Df(3L)vin7</i>	0920/16	P-pA	70D4-5	
0087/02	P-pA	68F	<i>Df(3R)crbS87-5</i>	0442/05	pA-A	70D4-7	
1472/07	A	68F and 71D	<i>Df(3L)vin7</i>	0430/08	L2	70D4-7	<i>Df(3R)by62</i>
0653/03	A	69B and 85E1-5		0938/06	E	70E	
1105/01	pA	69C		0446/12	A	70E	
0418/32	A	69C		0442/15	A	70E	
				1196/01	pA	70E	
				1379/08	L2	70E	

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Line	Lethal phase	Location	Deficiency	Line	Lethal phase	Location	Deficiency
0183/01	L3	70E	<i>Df(3R)T61</i>	0014/05	A	74F	
1190/05	L2	70F		1034/08	pA	75A	
1443/01	L3	70F		0017/17	pA	75B	
1464/11	A	70F		0269/05	A	75B	
0024/01	E-L1	70F	<i>Df(3L)ri-79c</i>	0343/05	P	75B	
0006/16	L2	70F	<i>Df(3R)T61 and Df(3R)P14</i>	0241/35	P	75B	
1479/04	E	71A	<i>Df(3R)kar-Sz28</i>	1460/04	pA	75B	
0749/13	pA-A	71B		0439/01	pA-A	75B	
0184/05	pP	71B	<i>Df(3L)Brd-12</i>	0194/18	pA-A	75B	
0277/07	L3-pP	71B	<i>Df(3L)Brd-12</i>	0569/06	A	75B	
1340/12	L2	71B	<i>Df(3L)GN50</i>	0920/11	Viable	75B	
0613/01	pA-A	71B1-3		0666/20	E	75B	
0968/05	E	71D	<i>Df(3L)Brd-12</i>	0456/08	A	75B	
0222/05	E	71D	<i>Df(3R)by62</i>	0855/05	A	75B	
0472/08	A	71E		0448/23	L3	75B	
1065/03	E	72B		0439/34	E	75B	
0626/11	E	72B	<i>Df(3L)st-f13</i>	1470/06	E	75B	
0805/03	pA-A	72B and 87C1-4		1001/08	E	75B	
0953/04	pP-P	72C		0232/04	P-pA	75B	<i>Df(3L)8IK19</i>
0413/15	L1	72C	<i>Df(3L)cat and Df(3L)st-f13</i>	0001/07	L2	75B	<i>Df(3L)W10</i>
0262/03	P	72F		1450/06	L3	75B	<i>Df(3L)W10</i>
0262/22	L3-pP	72F		0143/27	pA	75B	<i>Df(3L)W10</i>
0262/20	L3-pP	72F		0653/12	P-pA	75B	<i>Df(3R)by62</i>
1466/04	P	72F	<i>Df(3L)cat</i>	0351/01	E-L1	75B	<i>Df(3R)E307</i>
1232/08	E	72F	<i>Df(3L)st-f13</i>	0170/14	P-pA	75B	<i>Df(3R)T61</i>
0019/25	A	73A		0502/17	A	75B and 76C1-3	
0876/02	A	73A		1084/06	A	75B and 99A1-2	
0235/49	P	73A	<i>Df(3L)st-f13</i>	1022/11	pA	75B1-7	
1282/02	E	73A	<i>Df(3L)st-f13</i>	1368/08	E	75B11-C4	
0424/20	L3-pP	73A	<i>Df(3L)st-f13</i>	0145/11	Viable	75C	
0222/42	E	73B		0952/04	P-pA	75C	
1002/02	E	73B	<i>Df(3R)A117der21</i>	0040/21	pA-A	75C	
1120/14	A	73B and 79D		0523/19	A	75C	
1035/05	E	73B and 95C1-2		1152/14	pP	75C	<i>Df(3L)cat</i>
0736/15	P	73C	<i>Df(3L)8IK19</i>	1164/15	P	75C	<i>Df(3L)cat</i>
0479/06	E	73C	<i>Df(3L)8IK19</i>	1382/10	L3	75C	<i>Df(3L)cat</i>
0951/08	P	73D		1040/02	L2	75C	<i>Df(3R)by62</i>
0893/03	E	73E		0716/08	E	75C	<i>Df(3R)kar-Sz28</i>
1354/09	A	73E		0730/02	L3	75C and 75C	<i>Df(3L)cat</i>
0549/10	E	73E		0006/25	L1	75C1-2	<i>Df(3R)T61</i>
1314/04	A	73E and 83C1-3		1132/04	pA	75C1-4 and 85D6-12	<i>Df(3L)HR119</i>
1219/10	pA-A	73E and 84D1-5		1117/03	A	75D	
1372/08	E	73F and 100D		0951/09	E	75D	
1185/02	A	74A and 89B	<i>Df(3R)sbd26</i>	0882/05	E	75D	
0237/33	A	74B		1111/05	A	75D	<i>Df(3L)cat</i>
1031/14	P-pA	74B		0238/25	P	75D	<i>Df(3L)HR119</i>
0450/02	pA	74B and 91F10-13	<i>Df(3L)ri-79c</i>	1446/07	E	75D	<i>Df(3R)by62</i>
0902/14	pA-A	74C		1014/13	L2	75D	<i>Df(3L)cat</i>
0966/11	pA-A	74C		0483/01	P	75D, 90C	<i>Df(3L)cat</i>
0700/16	E	74C		0088/06	pA-A	75D4-8	
0520/01	pA-A	74C		0273/18	A	75E	
0415/06	P-pA	74D		0660/03	pA-A	75E	
0910/09	E	74D		0355/05	E	75E	<i>Df(3R)by62</i>
1039/09	A	74EF		0238/20	P	75E1-3	
0268/21	A	74F		0680/16	A	76A1-4	
				0524/01	pP-P	76A1-4	

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Line	Lethal phase	Location	Deficiency	Line	Lethal phase	Location	Deficiency
1135/09	E	76B	<i>Df(3R)T61</i>	0066/15	A	79D	
0703/10	A	76B1-3		1338/07	pA-A	79D and 83B6-9	
0474/31	A	76B1-5 and 99F1-3		0098/05	E-L1	79D and on X	<i>Df(3R)I26C</i>
0832/07	E	76B5-11		0681/11	L3	79D1-2	
0443/09	E	76C1-4	<i>Df(3R)E307</i>	0097/20	E	79E	
0669/17	E	76C4-6	<i>Df(3R)T61</i>	0003/06	A	79E	
0006/06	L1	76D		0001/22	A	79E	
0934/11	E	76D		0286/14	A	79E1-2	
1306/10	A	76D		0477/19	A	79E5-8	
0854/01	L2	76D		0446/23	A	79E5-8	
1457/09	L1	76D		0252/55	A	79F	
0755/15	pA-A	76D	<i>Df(3R)by62</i>	1393/08	A	80A and 88B5-9	
0237/23	A	76D1-4		0852/08	pA-A	82A	
1338/04	A	76F		0483/18	E	82A	
1338/01	A	76F		0479/16	L3-pP	82A	
1460/06	A	77A3-4		1343/04	A	82A	
0686/01	L1	77B		1278/16	A	82A and 91A3-6	
0256/04	pP-P	77B		0396/06	pA-A	82B	
0836/06	E	77B1-2		1280/10	A	82B	
1324/08	P	77B3-7		1436/09	A	82B	
0271/14	A	77C		0671/09	L2	82B	<i>Df(3R)by62</i>
1323/01	A	77C		0223/78	P-pA	82C	
1027/02	E	77C, 88A1-3, 89A1-4		0836/03	A	82C	
0396/05	A	77E1-4 and 84F	<i>Df(3R)crbS87-5</i>	0837/05	pA	82C	
1428/03	L1	77F	<i>Df(3R)E307</i>	1224/07	P	82C	<i>Df(3L)cat</i>
1254/14	E	78A		0918/01	P	82C1-3	
0309/01	E	78A		1300/08	E	82C1-3	<i>Df(3R)P14</i>
1428/14	E	78A	<i>Df(3R)by62</i>	0422/03	A	82D	
0006/27	E	78A1-3	<i>Df(3R)T61 and Df(3R)P14</i>	0422/05	A	82D	
0460/20	pA	78A1-4		1373/04	E	82D and on 2L	<i>Df(3R)I26C</i>
0651/04	A	78A2-5		0019/01	A	82D1-4 and 86D3-6	
0273/02	pA-A	78A4-7 and 92B1-5		0769/03	A	82D1-5	
0484/14	pA	78B		0919/04	E	82E	
0513/11	pA-A	78C		1044/16	P-Pa	82E	<i>Df(3L)cat</i>
1111/16	A	78C		0517/01	pP-P	82E	
0513/09	Viable	78C		0506/20	P	82E and 83D and 88C	
1020/12	A	78C		0474/18	P	82E1-3	
0686/07	A	78C		0672/05	pA-A	82E4-8	
0446/01	pA-A	78C		0321/11	A	82F	
0475/23	A	78C and 89A		0188/01	A	82F	
0838/05	P-pA	78D and 98C		0110/01	A	82F and 89C	<i>Df(3R)sbd26 and Df(3L)ri-79c</i>
0448/35	A	78D1-4		0424/05	A	82F and 97C	
1083/13	E	78E		0849/10	E	82F1-2 and 84F11-16	
0496/10	A	78E		1216/12	P-pA	82F1-2 and 86E10-15	
0554/18	pA-A	78F		0014/07	pA-A	82F1-2 and 87D10-14	
0721/14	A	79A1-4		0241/51	A	82F1-3	
0472/22	A	79B		0236/39	A	82F1-3	
0707/17	A	79B		0303/04	pA-A	82F3-6	
1432/06	A	79B		1471/04	pA-A	82F3-7	<i>Df(3R)by62</i>
0549/08	pA	79B and 80C		0443/06	A	82F8-9	
0632/07	A	79B1-3 and 93D		0077/08	pA-A	82F8-9	
0990/06	A	79B3		0529/08	L2	83A	<i>Df(3L)HR119</i>
0898/14	P-pA	79C					
0099/19	A	79C					
0036/01	pA-A	79C					

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Line	Lethal phase	Location	Deficiency	Line	Lethal phase	Location	Deficiency
0954/14	P	83A1-2		0976/13	E	85A1-5	
0965/11	E	83A5-9		0770/11	A	85A1-6 and 87C4-8	
0703/16	pA	83B		1267/11	A	85B	
0967/03	P	83B		0273/35	L3	85B	
0472/14	A	83B		0239/41	pA	85B	<i>Df(3L)Brd-12 and Df(3L)ri-79c</i>
1312/16	A	83B		1287/11	A	85B4-9	
1017/10	P	83B		0751/01	pA-A	85B6-9	
0114/06	pA-A	83B		0486/06	E	85C	
0428/34	L2	83B	<i>Df(3L)cat</i>	0705/03	pA-A	85C	
0480/15	L2	83B	<i>Df(3L)ri-79c</i>	1293/09	E	85C	
0166/03	E-L1	83B	<i>Df(3R)A117der21</i>	0982/02	P	85C	
0001/01	E	83B	<i>Df(3R)A117der21</i>	1315/05	E	85C	
0324/14	A	83B3-7		0425/32	A	85C10-13 and 98D4-7	
0923/02	pA-A	83B4-8		1261/04	A	85C7-11	
0577/06	P	83C		1380/06	A	85C8-12 and 86E9-15	
0208/02	L1	83C		0328/12	E	85C9-10	
0843/08	E	83C		1056/11	A	85C9-13	
0115/13	E	83C	<i>Df(3L)ri-79c</i>	0971/15	E	85C9-15	
1344/15	L3	83C1-3		1267/05	pA-A	85D	
0958/12	pA	83D		0561/13	E	85D	<i>Df(3R)by62</i>
0082/06	L1	83D		0420/08	P-pA	85D1-2	
0793/04	E	83D		0899/04	pA	85D1-2	
0773/01	P-pA	83F		0492/05	pA-A	85D1-2, 91F8-13, 98B1-5	
0486/19	E	83F		0499/06	P-pA-A	85D1-3 and 85D20-25	<i>Df(3R)P14</i>
0487/16	P-pA	83F		1448/02	E	85D1-3, 90C1-3, 100D	
0732/14	A	83F		0236/34	A	85D1-4	
0414/05	A	83F		0597/05	A	85D1-4 and 87E4-12	
0414/15	A	83F		1134/03	A	85D1-5	
0303/02	P	83F	<i>Df(3R)E307</i>	0405/06	A	85D1-5	
0708/06	E	83F and 85F	<i>Df(3R)by62</i>	0936/14	L3	85D1-5	
0957/14	E	84A		1285/01	P-pA	85D1-5	<i>Df(3R)T61</i>
0278/21	A	84A1-3		0545/07	L3	85D1-8	<i>Df(3R)P14</i>
0935/01	L2	84A3-6		0428/25	pA	85D10-12	
1474/06	P	84B3-6	<i>Df(3L)cat</i>	1228/04	E	85D12-20	
0578/09	E	84C and 94A		1243/10	P-pA-A	85D14-18	
0973/01	L3	84D		0264/09	A	85D14-18 and 88C	<i>Df(3R)by62</i>
0074/03	E	84D	<i>Df(3R)126C</i>	1177/08	P-pA	85D16-20	
1274/16	pA-A	84D1-4		0264/30	A	85D16-20	<i>Df(3R)by62</i>
1451/10	A	84D1-7		0662/08	pA-A	85D17-22	<i>Df(3R)by62</i>
0501/15	pA	84D4-8		0672/06	A	85D17-23	
0252/40	A	84E		1457/01	A	85D20-25	
0965/16	L3	84E		1440/02	L2	85D20-27 and 73B	
0276/04	E	84E10-13	<i>Df(3R)T61</i>	1053/07	E	85D21-27	<i>Df(3R)by62</i>
0585/07	A	84E8-12		1446/08	A	85D9-14	
0404/28	A	84E9-13		0701/06	A	85E	
0238/03	E	84F		0423/13	Viable	85E	
1351/08	E	84F		0904/11	E	85E	
0110/28	E-L1	84F		1175/05	E	85E	<i>Df(3R)by62</i>
0587/12	E	84F		0363/02	P	85E	<i>Df(3R)by62</i>
0985/13	E	84F	<i>Df(3R)by62</i>	0079/34	E	85E	<i>Df(3R)by62</i>
0887/12	E	84F					
0791/01	A	84F12-16					
0961/01	E	84F13-16 and 90E					
0241/06	P-pA	84F2-4					
0609/14	A	85A					
0282/06	L3-pP	85A					
0258/07	E	85A and 95F	<i>Df(3R)crbS87-5</i>				

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Line	Lethal phase	Location	Deficiency	Line	Lethal phase	Location	Deficiency
0265/43	A	85E1-5		0671/02	E	86E2-5	<i>Df(3R)T61</i>
0484/12	A	85E1-5	<i>Df(3L)h-i22</i>	1292/17	A	86E6-8	
0392/06	E	85E1-7		0096/47	E	86E9-11	
0335/13	pA-A	85E1-7	<i>Df(3R)by62</i>	0762/01	A	86E9-12	
0451/09	E	85E309	<i>Df(3R)T61 and Df(3R)karSz13</i>	1419/14	A	86E9-13, 87C1-3 and 2L	
0481/03	pA	85E6-10	<i>Df(3R)by62</i>	1019/02	E	86F	
0261/15	E	85E8-15 and 22A	<i>Df(3R)by62</i>	0501/16	L3	86F	
0346/02	A	85F		0110/46	pP	86F	
0192/03	pP-P	85F	<i>Df(3R)by62</i>	0483/19	A	86F	
1330/08	E	85F	<i>Df(3R)karSz28</i>	0493/14	E	86F and 100A	<i>Df(3L)ri-79c</i>
0618/05	pA	85F11-16	<i>Df(3R)by62</i>	0053/02	P	86F and 75B	
1034/02	E	85F13-16	<i>Df(3R)karSz28</i>	1250/12	pP-P	86F3-10	
0281/08	P	85F9-16	<i>Df(3R)by62</i>	1132/08	L3	86F3-6	
1417/15	L3	85F9-16	<i>Df(3R)by62</i>	1208/13	L2	86F3-9 and 87C1-3	<i>Df(3R)E307</i>
0229/11	L2	85F9-16	<i>Df(3R)by62</i>	0440/06	A	86F4-9	
1232/13	pA	85F9-16	<i>Df(3R)by62</i>	1442/01	pA-A	86F4-9	
1426/01	pA	85F9-16	<i>Df(3R)by62</i>	0504/07	E	86F6-10 and 88E5-10	<i>Df(3R)T61</i>
1026/09	P-pA	85F9-16	<i>Df(3R)by62</i>	0549/07	E-L1	87A1-2	<i>Df(3R)T61 and Df(3R)P21</i>
1003/03	P	85F9-16	<i>Df(3R)by62</i>	1261/13	pA-A	87A6-10 and 94B5-10	
0955/15	E	85F9-16	<i>Df(3R)by62</i>	0861/07	E	87B	
1367/03	pA	85F9-16	<i>Df(3R)by62</i>	1353/01	Viable	87B	
0019/06	P	85F9-16 and 65D	<i>Df(3R)by62</i>	0444/02	P	87B1-2	<i>Df(3R)E229</i>
1145/13	E	85F9-16 and 70F	<i>Df(3R)by62</i>	0114/20	E	87B1-5	<i>Df(3R)E307</i>
1437/02	E	86A		0598/01	E	87B1-5	<i>Df(3R)E307</i>
0445/05	A	86A		0106/18	pA-A	87B1-7	
0229/05	A	86A and 91F		0288/13	P-pA-A	87B10-15	
1366/13	pA	86A1-2		0842/04	L2	87B10-15	
1248/02	pA-A	86A1-5		0005/12	E	87B3-5	<i>Df(3R)E307</i>
1207/10	pA-A	86B		0042/01	E	87B3-6	<i>Df(3R)E307</i>
0038/31	A	86B	<i>Df(3R)sbd26</i>	1107/09	A	87B4-8	
0958/08	A	86B and 98A		0109/17	pA-A	87B5-9	
0234/34	pA-A	86C and 89B	<i>Df(3R)sbd26</i>	1392/13	A	87C	
1422/04	L1	86C1-6		0516/01	A	87C1-3	
0778/01	E	86C1-6		1084/08	A	87C1-3 and 100E	
0324/01	E	86C1-6		0742/05	A	87C5-9	<i>Df(3R)karSz28</i>
1323/07	E	86C1-6	<i>Df(3L)fz-GF3b</i>	0030/06	A	87D1-2	
0972/01	L2	86C9-15		0369/09	A	87D1-5	
0337/02	E-L1	86D	<i>Df(3L)fz-GF3b</i>	1250/06	A	87D1-5	
0944/05	P-pA	86D1-2		0256/16	L3	87D6-14	
0006/24	L3	86D1-3	<i>Df(3R)T61 and Df(3R)P14</i>	0221/22	E	87D9-14 and 30A	<i>Df(3R)by62</i>
0696/05	E	86D1-5	<i>DF(3R)T61</i>	1083/10	E	87E	
0979/07	L3	86E	<i>Df(3R)crbS87-5</i>	0616/17	L2	87E	<i>Df(3R)126C</i>
0989/01	A	86E1-2		1131/05	L3	87E	<i>Df(3R)126C</i>
0420/18	pA-A	85E1-3		0263/16	L1	87E	<i>Df(3R)karSz28</i>
0585/13	A	86E1-3		1258/16	E	87E1-3	<i>Df(3R)P14 and Df(3R)karSz28</i>
0563/18	A	86E1-3		0081/31	A	87E5-10 and on 2R	
0763/13	E	86E1-3	<i>Df(3R)T61</i>	1437/04	A	87E7-12	<i>Df(3R)126C</i>
0058/18	L2	86E1-5		1018/04	L2	87E8-12	<i>Df(3R)126C</i>
0441/16	pA	86E1-5	<i>Df(3R)T61</i>	0708/15	L2	87F	
0664/07	L2	86E1-5	<i>Df(3R)T61</i>	0110/04	pA-A	87F	
0095/12	A	86E1-6		1295/10	E	87F	<i>Df(3R)126C</i>
0943/10	P-pA	86E1-6		1064/14	pA	87F	<i>Df(3R)126C</i>
0320/10	A	86E1-6					
0179/09	pA	86E14-20	<i>Df(3R)T61</i>				
0300/03	P-pA	86E14-20	<i>Df(3R)T61</i>				

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Line	Lethal phase	Location	Deficiency	Line	Lethal phase	Location	Deficiency
0299/10	P	87F	<i>Df(3R)126C</i>	0941/05	E	88F1-2	
0086/14	E	87F	<i>Df(3R)126c</i>	0839/02	E	88F1-2	
1303/13	E	87F1-3	<i>Df(3R)E307</i>	0055/15	pA	88F1-2	
1041/01	E	87F1-8		0482/19	A	88F1-3	
0110/41	pA-A	84F10-15		0458/22	A	88F1-3	
1029/10	P	87F10-15	<i>Df(3R)126C</i>	1190/16	A	88F1-3	
0904/17	E	87F3-10		0701/16	A	88F6-8	
0239/43	pA-A	88A		0897/07	E	89A	
1466/07	L3	88A		0680/02	P	89A	<i>Df(3R)crbS87-5</i>
1250/11	A	88A and 89B		1306/08	A	89A3-10	
0848/05	E	88A1-2		0571/14	A	89B	
0869/09	E	88A1-2		0967/05	E	89B	
1012/06	E	88A1-2	<i>Df(3L)GN50</i>	0234/50	pA	89B	
0785/14	pA-A	88A1-5		1439/16		89B	
1388/15	P	88A1-5		0484/06		89B	
0596/13	A	88A4-8 and 94A1-5		0073/59	A	89B	
0413/16	P	88A6-12	<i>Df(3L)cat</i>	0284/04	L3-pP	89B	
1186/02	E	88B	<i>Df(3L)ZP-1</i>	0842/10	pA	89B	
0927/08	L3	88B1-2		0717/10	A	89B	<i>Df(3R)sbd26</i>
0510/07	E	88B1-2 and 100F		0088/40	P-pA	89B	<i>Df(3R)sbd26</i>
0910/14	E	88B1-3		0234/47	A	89B	<i>Df(3R)sbd26</i>
1323/04	P	88B1-3	<i>Df(3)cat</i>	0651/03	P-pA	89B	<i>Df(3R)sbd26</i>
1327/03	A	88B1-4		0582/09	A	89B and 100D	
1479/13	A	88B3-6		1193/07	E	89B and 96A	<i>Df(3R)crbS87-5</i>
0007/21	A	88B3-6		0053/10	pP	89B1-5	
1237/13	E	88C		1310/10	A	89B10-15	
0940/03	E	88C		1066/05	A	89B10-15	<i>Df(3R)sbd26</i>
1262/01	E	88C		0930/08	A	89B11-16	
1054/07	E	88C		0989/16	P	89B14-20	<i>Df(3R)sbd26</i>
1297/17	E	88C		1343/11	A	89B15-20	
1104/08	E	88C		0037/04	A	89B16-20	<i>Df(3R)sbd26</i>
0016/20	L1	88C	<i>Df(3R)P14</i>	0248/34	L1	89B16-22	<i>Df(3R)by62</i>
0222/31	P-pA-A	88C and 2R		0414/11		89B4-15	
0837/10	pA-A	88C and 93B1-2		1275/08	A	89B5-10	
0447/38	A	88D		0273/44	A	89B5-10	<i>Df(3R)sbd26</i>
0447/29	A	88D		1463/15	A	89B6-10	
0741/03	P-pA	88D		0201/14	A	89B8-14	<i>Df(3R)sbd26</i>
1473/13	A	88D		1042/10	L2	89B8-15	<i>Df(3L)ZP-1</i>
0864/05	L3	88D		0521/11	pA	89D	
0494/05	E	88D	<i>Df(3R)P14</i>	0607/08	A	89D	
0205/14	pA	88D and 100B	<i>Df(3R)A117der21</i>	0607/11	A	89D1-3	<i>Df(3R)sbd26</i>
1121/10	E	88D1-4		0961/08	P-pA	89E	
0843/05	E	88E		0501/10	pA	89E	
1023/03	A	88E		1480/11	pA	89E5-11	
1003/09	E	88E		0422/02	A	89E9-13	
0078/18	E-L1	88E	<i>Df(3R)P14</i>	0527/13	A	89F	
0926/11	E	88E and 92A		1429/10	E	90A	<i>Df(3R)T61</i>
1224/13	E	88E1-2	<i>Df(3L)cat</i>	0965/12	E	90B	
0942/04	E	88E1-4		0746/06	pA-A	90B1-3 and 99F1-3	
1227/09	pP	88E1-4	<i>Df(3R)T61</i>	1422/14	A	90B4-8	
0775/15	A	88E3-4		0379/01	A	90C	
0091/13	E	88E5-10	<i>Df(3R)kar-Sz28</i>	1448/12	E	90C and 100D	
0911/04	E	88E5-10 and 2R(52B)		0477/16	L3-pP	90C5-10	
0850/05	E	88F		0881/13	E	90C5-10 and 100E	
1110/04	E	88F		1406/12	E	90D	
0424/12	A	88F		0225/27	P-pA	90D	
				1026/11	E	90D	

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Line	Lethal phase	Location	Deficiency	Line	Lethal phase	Location	Deficiency
0377/03	pA-A	90D		0495/20	E	92A	
1003/05	E	90D		1423/11	E	92A	
1381/02	E	90D		1440/11	E	92A	
0550/09	E	90D	<i>Df(3L)h-i22</i>	1104/16	P-pA	92A	
1445/07	pA-A	90D	<i>Df(3L)HR119</i>	0246/39	E	92A	<i>Df(3L)ri-79c</i>
0223/48	E-L1	90D	<i>Df(3R)P14</i>	1155/10	E	92A	<i>Df(3L)ri-79c</i>
0452/30	E	90D	<i>Df(3R)P14</i>	0260/20	E-L1	92A and 100D	<i>Df(3L)ri-79c</i>
1417/10	E	90D	<i>Df(3R)T61</i>	0861/02	E	92A1-2	
0977/09	E	90D and 61D		0663/17	E	92A1-3	
0455/07	E	90D1-4	<i>Df(3R)P14 and Df(3R)T61</i>	0861/12	E	92A1-3	
0986/14	E	90D3-6		0903/02	E	92B	
0113/02	E	90E		0573/10	A	92B	
1456/13	A	90E1-3		1023/15	pA-A	92B1-3	
0236/37	E	90E1-3	<i>Df(3R)P14</i>	0729/12	P-pA-A	92B1-3	
0324/12	E-L1	90E1-3	<i>Df(3R)P14</i>	0985/16	E	92B1-3	
1316/02	E	90E3-6	<i>Df(3R)T61</i>	0094/18	A	92B1-3	
0967/13	L3	90F		0969/01	P-pA	92B1-3	<i>Df(3L)Brd-12</i>
0763/04	A	90F		1407/13	A	92B1-3	<i>Df(3L)HR119</i>
0255/07	A	90F		1039/14	L3	92C	
0019/17	P	90F		1167/13	E	92C	<i>Df(3R)T61</i>
0981/05	pA-A	91A and 91A		0487/06	pA	92E	
1419/08	A	91A3-6		0434/18	A	92E10-15	
0747/06	pA-A	91A4-7		0244/09	E	92E4-9 and 2R	<i>Df(3R)T61</i>
0715/01	pA-A	91A4-7	<i>Df(3R)P14</i>	0434/20	A	92E8-11	
1159/09	pA-A	91A4-8		0249/12	pA	92E8-14	
0245/06	A	91B		0241/08	pA	92E8-14	
0946/03	E	91B		0675/01	A	92F1-2	
1005/02	L3	91B1-5		0300/09	pA	93A1-5	
0073/02	E	91B1-6		0835/02	E	93B1-2	
0885/01	E	91B-2 and 99D1-2		1309/10	E	93B1-4	
1482/13	pA-A	91B4-6		1389/01	pP-P-pA	93B4-8	
0281/02	pA-A	91C		0264/20	A	93B9-13	
1077/07	A	91C		0803/01	A	93C	
0970/02	E	91D		0127/19	L3	93C	
1077/01	A	91D		0050/42	L3	93C	
0673/04	L3	91F		0448/28	A	93C1-5	
0229/30	A	91F		0937/05	E	93D	
0584/07	pA-A	91F		0571/15	L3	93D6-10	
0701/03	E	91F		0297/01	L1	93E	<i>Df(3R)by61 and Df(3R)P14</i>
1108/12	A	91F		0524/13	Viable	93E3-5	
1386/13	E	91F	<i>Df(3L)HR119</i>	0525/01	Viable	93E3-7	
0929/02	E	91F1-2		0623/14	A	93E6-11	
0775/13	A	91F1-5		0623/10	A	93E6-11	
0247/06	E	91F1-5		1023/12	E	93E8-11	
1144/14	P-pA-A	91F1-8		0824/14	A	93EF	<i>Df(3R)by62</i>
0807/07	P	91F1-8	<i>Df(3L)W10</i>	0868/02	E	93F	
0985/02	E	91F3-5, 68C1-5, 71B		1117/04	L1	93F11-14	<i>Df(3R)by62</i>
1125/17	pA	91F3-8	<i>Df(3R)T61</i>	0446/32	A	93F3-8	
0426/22	pA-A	91F8-12		0924/05	E	94A	
0167/04	pA-A	91F8-13		0913/06	L2	94A	
1111/10	E	91F9-13	<i>Df(3R)kar-Sz28</i>	1017/12	E	94A	
1304/03	E	92A		0692/06	L2	94A1-4	<i>Df(3R)by62</i>
1053/14	E	92A		0263/32	A	94A5-10	
1119/09	E	92A		1302/04	A	94A8-16	
1485/04	E	92A		0734/03	A	94B	
				1298/03	A	94B1-4	

APPENDIX A

Continued

Line	Lethal phase	Location	Deficiency	Line	Lethal phase	Location	Deficiency
0734/14	A	94C		1045/09	E	97D	<i>Df(3L)ri-79c</i>
1343/06	E	94D	<i>Df(3L)st-f13</i>	0188/09	Viable	97E1-6	
0025/01	A	94E		0715/02	pA-A	97F1-3	
0149/17	P	94E		0485/18	pA-A	98A1-5	
0513/02	A	94F		0318/07	E	98A5-10	
0998/12	P	94F		0857/06	E	98B	
0122/25	L1	94F	<i>Df(3R)by62</i>	0957/08	E	98B1-2	
0513/03	A	94F1-3		0430/05	P-pA	98B5-8	<i>Df(3L)cat</i>
0123/09	E-L1	94F1-3		0225/29	A	98C	
1015/04	pA-A	95B		0031/08	A	98C	
0924/16	E	95B		1041/04	pA	98D	
0643/16	L2	95B	<i>Df(3L)W10</i>	0595/08	E	98D	
1002/01	E	95B1-2		1351/07	pA	98D3-7	
1004/09	pA	95C	<i>Df(3R)cnbS87-5</i>	0398/07	A	98D4-7	
0490/09	P-pA	95C1-8		1409/01	L2	98D4-7	
0718/14	A	95C6-9		1013/10	pA	98E1-2	
0700/05	A	95D		0962/07	pA-A	98F	
0383/07	A	95D		0151/02	E	98F	<i>Df(3R)A117der21</i>
0903/03	E	95D1-2		0677/12	P	98F	<i>Df(3R)cnbS87-5 and Df(3R)T61</i>
1261/10	pA-A	95D1-6		0844/02	L2	98F1-3	
0289/29	A	95E		0444/22	L1	98F1-5	
1381/11	A	95E1-3		0852/03	A	98F1-5	
1381/12	A	95E1-3		0899/14	E	98F10-14	
1381/07	A	95E1-3 and 100D		0730/13	E	99A	
0001/08	A	95E5-8		0439/22	E	99A	
0745/10	L3	95F		0980/06	E	99A	
0509/11	E	95F	<i>Df(3R)cnbS87-5</i>	1089/08	E	99A	
0032/20	E	95F	<i>Df(3R)cnbS87-5</i>	0853/01	P	99A	
1355/06	E	95F	<i>Df(3R)cnbS87-5</i>	0245/03	E	99A	
1384/04	A	95F	<i>Df(3R)cnbS87-5</i>	0224/06	E	99A1-4	
0104/09	pA-A	95F	<i>Df(3R)cnbS87-5</i>	0079/02	L3	99A1-4	
0581/04	E	95F	<i>Df(3R)cnbS87-5</i>	0850/09	L3	99A1-6	
0156/01	E	95F and 90D	<i>Df(3R)cnbS87-5</i>	1060/06	L3	99A1-8 and 100F	
0903/08	A	95F1-2		0200/04	A	99B7-11	
0248/36	L2	95F10-15	<i>Df(3R)cnbS87-5</i>	0529/04	pA-A	99D	
0016/04	E	95F10-15	<i>Df(3R)cnbS87-5</i>	1036/03	A	99D	
0797/09	E	95F10-15	<i>Df(3R)cnbS87-5</i>	0829/08	P	99D	<i>Df(3L)Brd-12</i>
1426/03	L2	95F12-15 and 94B	<i>Df(3R)cnbS87-5</i>	0479/09	A	99D1-2	
0053/43	P	95F3-10		0442/30	E	99D3-9	
0672/10	P-pA-A	95F5-10		1037/05	L2	99E	
1029/08	L3	95F7-9; 96A17-18		1196/08	A	99E	
1124/11	A	96A11-16		1305/09	A	99F	
0924/15	E	96A16-20		0282/18	E	99F	
0417/06	E	96A3-16	<i>Df(3R)cnbS87-5</i>	1384/15	E	99F	<i>Df(3L)HR119</i>
1337/05	L1	96A8-14	<i>Df(3R)cnbS87-5</i>	0231/41	L2	99F	<i>Df(3R)by62</i>
0183/10	A	96B		0834/07	L1	99F1-2	
0444/37	A	96B and 100F		0830/05	P-pA	99F1-2	
1233/04	pA-A	96C		0904/05	pA-A	99F1-2	
1100/08	L2	96C	<i>Df(3R)by62</i>	1049/13	A	99F1-2	
0472/12	pA	96C7-9		1049/03	L3-pP	99F1-3	<i>Df(3L)GN50</i>
0772/13	pA-A	96F10-14		0143/08	L1	99F1-4	<i>Df(3L)GN50</i>
0638/18	A	96F11-14		1081/16	P-pA	99F1-4	
1439/07	L3-pP-P	96F11-14		0050/29	E-L1	99F1-4	<i>Df(3L)81K19</i>
0638/11	A	96F8-11		0946/15	E	99F6-11	
0099/11	A	96F8-12		1277/04	E	100A	<i>Df(3L)GN50</i>
1006/04	E	97A		1274/08	pA	100A	<i>Df(3R)A117der21</i>
1396/14	E	97D					

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Line	Lethal phase	Location	Deficiency	Line	Lethal phase	Location	Deficiency
0538/13	L2	100B	<i>Df(3R)A117der21</i>	1475/06	E		<i>Df(3L)81K19</i>
0353/13	P	100B	<i>Df(3R)A117der21</i>	0081/36	L1		<i>Df(3L)AC1</i>
0553/10	E	100B	<i>Df(3R)A117der21</i>	0238/09	pA		<i>Df(3L)AC1</i>
0990/13	E	100B	<i>Df(3R)A117der21</i>	0023/02	pA		<i>Df(3L)AC1</i>
0915/10	P	100B	<i>Df(3R)A117der21</i>	0123/11	E		<i>Df(3L)AC1</i>
0812/02	L3-pP	100B	<i>Df(3R)A117der21</i>	0252/47	E		<i>Df(3L)AC1</i>
0966/01	P-pA	100B	<i>Df(3R)A117der21</i>	0809/06	L3		<i>Df(3L)Brd-12</i>
0726/03	E	100B	<i>Df(3R)A117der21</i>	0448/05	L1		<i>Df(3L)Brd-12</i>
1447/01	E	100B	<i>Df(3R)A117der21</i>	0643/18	L3		<i>Df(3L)Brd-12</i>
0236/35	E	100B	<i>Df(3R)A117der21</i>	0974/08	E		<i>Df(3L)Brd-12</i>
1396/02	L3	100B1-5	<i>Df(3R)A117der21</i>	0906/05	E		<i>Df(3L)Brd-12</i>
1324/13	E	100B6-9	<i>Df(3R)A117der21</i>	0730/15	L3		<i>Df(3L)Brd-12</i>
0573/02	E	100C		0828/07	E		<i>Df(3L)Brd-12</i>
0250/25	E	100D		1446/06	E		<i>Df(3L)Brd-12</i>
0274/18	A	100D		1402/07	L2		<i>Df(3L)Brd-12</i>
1209/10	E	100D		0584/12	L2		<i>Df(3L)Brd-12</i>
1184/16	E	100D		0139/04	L3-pP		<i>Df(3L)Brd-12</i>
0082/24	pP-P	100D		1109/15	E		<i>Df(3L)Brd-12</i>
1260/10	E	100D		1480/16	L2		<i>Df(3L)Brd-12</i>
0702/07	E	100D		0594/03	P		<i>Df(3L)Brd-12</i>
1281/04	E	100D		0609/03	L2		<i>Df(3L)Brd-12</i>
1209/05	E	100D		0594/16	L2		<i>Df(3L)Brd-12</i>
1049/07	E	100D		0974/05	E		<i>Df(3L)Brd-12</i>
1391/01	L3	100D		0613/08	P		<i>Df(3L)Brd-12</i>
0037/17	E-L1	100D		1222/11	L2		<i>Df(3L)Brd-12</i>
1325/15	E	100D		0621/12	pA		<i>Df(3L)cat</i>
1418/06	E	100D		1469/01	P		<i>Df(3L)cat</i>
0770/09	A	100D		0612/07	E		<i>Df(3L)cat</i>
0438/31	E	100D		0610/02	P-pA		<i>Df(3L)cat</i>
1321/07	A	100D		0597/06	E		<i>Df(3L)cat</i>
1119/04	E	100D		1474/10	P		<i>Df(3L)cat</i>
1103/05	E	100D	<i>Df(3L)st-f13</i>	0428/24	L3		<i>Df(3L)cat</i>
0509/10	A	100E		0816/07	P-pA		<i>Df(3L)cat</i>
0261/11	A	100E		1165/12	P-pA		<i>Df(3L)cat</i>
0991/02	A	100E		0690/07	E		<i>Df(3L)cat</i>
1346/01	A	100E	<i>Df(3R)by62</i>	0238/26	L2		<i>Df(3L)cat</i>
0840/13	E	100E2-F		1323/12	P		<i>Df(3L)cat</i>
0243/14	P-pA	100F		1322/14	P		<i>Df(3L)cat</i>
0413/03	A	100F		1474/02	P		<i>Df(3L)cat</i>
0998/01	L2	100F		0238/16	P		<i>Df(3L)cat</i>
0952/14	L2	100F		0685/02	P-pA		<i>Df(3L)cat</i>
1344/08	A	100F1-3		0300/04	P-pA		<i>Df(3L)cat</i>
1353/05	A	100F3-5		0291/20	L3-pP-P-pA		<i>Df(3L)cat</i>
0919/12	E		<i>Df(3L)29A6</i>	0743/09	E		<i>Df(3L)cat</i>
0587/11	E		<i>Df(3L)29A6</i>	1226/12	E		<i>Df(3L)cat</i>
0589/11	E		<i>Df(3L)29A6</i>	0671/10	E		<i>Df(3L)cat</i>
0260/09	L2		<i>Df(3L)29A6</i>	0649/06	E		<i>Df(3L)fz-GF3b</i>
0147/08	P-pA		<i>Df(3L)81K19</i>	0009/03	L1		<i>Df(3L)HR119</i>
0115/24	L2		<i>Df(3L)81K19</i>	0686/09	pA		<i>Df(3L)HR119</i>
0682/12	E		<i>Df(3L)81K19</i>	1154/15	P-pA		<i>Df(3L)HR119</i>
0633/19	E		<i>Df(3L)81K19</i>	0034/05	P-pA		<i>Df(3L)ri-79c</i>
0383/05	pA		<i>Df(3L)81K19</i>	1345/05	E		<i>Df(3L)st-f13</i>
0700/06	E		<i>Df(3L)81K19</i>	0736/01	E		<i>Df(3L)st-f13</i>
0497/06	L2		<i>Df(3L)81K19</i>	0542/11	E		<i>Df(3L)st-f13</i>
0759/01	E		<i>Df(3L)81K19</i>	0643/01	E		<i>Df(3L)st-f13</i>
1471/14	E		<i>Df(3L)81K19</i>	0306/05	L2		<i>Df(3L)st-f13</i>
0270/05	P-pA		<i>Df(3L)81K19</i>	0573/06	L3		<i>Df(3L)st-f13</i>

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Line	Lethal phase	Location	Deficiency	Line	Lethal phase	Location	Deficiency
0901/01	L3-pP		<i>Df(3L)st-f13</i>	0608/04	L3		<i>Df(3R)by62</i>
0053/31	P		<i>Df(3L)st-f13</i>	0804/09	L2		<i>Df(3R)by62</i>
0030/03	E-L1		<i>Df(3L)st-f13</i>	0499/02	L2		<i>Df(3R)by62</i>
0489/15	E		<i>Df(3L)st-f13</i>	0426/29	L3		<i>Df(3R)by62</i>
0644/11	E		<i>Df(3L)st-f13</i>	0244/55	pP-P		<i>Df(3R)by62</i>
1103/03	E		<i>Df(3L)st-f13</i>	0885/13	E		<i>Df(3R)by62</i>
1225/01	E		<i>Df(3L)win7</i>	0485/07	E		<i>Df(3R)by62</i>
0150/12	L1		<i>Df(3L)win7</i>	0310/06	E		<i>Df(3R)by62</i>
1199/06	E		<i>Df(3L)win7</i>	0426/30	L2		<i>Df(3R)by62</i>
1473/10	P		<i>Df(3L)win7</i>	1199/08	pP		<i>Df(3R)by62</i>
0599/13	E		<i>Df(3L)win7</i>	0919/05	E		<i>Df(3R)by62</i>
0446/28	L3		<i>Df(3L)win7</i>	0430/29	L3		<i>Df(3R)by62</i>
0446/26	E		<i>Df(3L)win7</i>	0294/03	E		<i>Df(3R)by62</i>
1067/10	P-pA		<i>Df(3L)win7</i>	0273/13	E-L1		<i>Df(3R)by62</i>
0931/08	E		<i>Df(3L)win7</i>	0271/27	L1		<i>Df(3R)by62</i>
0245/33	L2		<i>Df(3L)win7</i>	0751/10	L2		<i>Df(3R)by62</i>
1197/15	E		<i>Df(3L)win7</i>	0263/26	L2		<i>Df(3R)by62</i>
0693/07	L3		<i>Df(3L)win7</i>	0262/26	E		<i>Df(3R)by62</i>
1482/14	P-pA		<i>Df(3L)win7</i>	0764/15	L2		<i>Df(3R)by62</i>
0770/08	P		<i>Df(3L)win7</i>	0323/03	L1		<i>Df(3R)by62</i>
0251/32	P-pA		<i>Df(3L)W10</i>	0660/17	E		<i>Df(3R)by62</i>
1118/16	L1		<i>Df(3L)W10</i>	1466/06	L2		<i>Df(3R)by62</i>
0970/01	E		<i>Df(3L)W10</i>	0258/32	A		<i>Df(3R)by62</i>
1411/10	E		<i>Df(3L)W10</i>	0248/38	L3		<i>Df(3R)by62</i>
1087/09	E		<i>Df(3L)W10</i>	0291/10	E-L1		<i>Df(3R)by62</i>
1118/02	E		<i>Df(3L)W10</i>	0230/13	L2		<i>Df(3R)by62</i>
0122/24	E		<i>Df(3L)W10</i>	0259/13	pP		<i>Df(3R)by62</i>
0661/21	E		<i>Df(3L)W10</i>	0258/06	E-L1		<i>Df(3R)by62</i>
0263/09	pA		<i>Df(3L)W10</i>	0339/03	E-L1		<i>Df(3R)by62</i>
0082/23	E		<i>Df(3L)W10</i>	0233/09	E		<i>Df(3R)by62</i>
1186/08	A		<i>Df(3L)ZP-1</i>	0947/13	E		<i>Df(3R)crbS87-5</i>
1435/17	L1		<i>Df(3L)ZP-1</i>	0632/18	E		<i>Df(3R)crbS87-5</i>
1179/02			<i>Df(3L)ZP-1</i>	0509/20	E		<i>Df(3R)crbS87-5</i>
1179/13	A		<i>Df(3L)ZP-1</i>	1467/05	E		<i>Df(3R)E307</i>
0079/15	E		<i>Df(3R)A117der21</i>	0234/26	E		<i>Df(3R)P14</i>
0679/15	E		<i>Df(3R)by62</i>	0288/30			<i>Df(3R)P14</i>
1329/07	E		<i>Df(3R)by62</i>	1251/04	E		<i>Df(3R)P14</i>
0239/38	E		<i>Df(3R)by62</i>	1367/08	E		<i>Df(3R)P14</i>
1413/09	L3		<i>Df(3R)by62</i>	0456/02	E		<i>Df(3R)P14</i>
0530/11	L2		<i>Df(3R)by62</i>	0698/05	E		<i>Df(3R)P14</i>
0602/03	L3		<i>Df(3R)by62</i>	1357/07	E		<i>Df(3R)P14</i>
0619/15	E		<i>Df(3R)by62</i>	0283/17	E		<i>Df(3R)P14</i>
0668/13	L2		<i>Df(3R)by62</i>	0283/10	E		<i>Df(3R)P14</i>
0287/07	E		<i>Df(3R)by62</i>	0727/03	E		<i>Df(3R)P14</i>
1108/15	L2		<i>Df(3R)by62</i>	0321/10	E		<i>Df(3R)P14</i>
0379/06	L2		<i>Df(3R)by62</i>	1293/10	E		<i>Df(3R)P14</i>
0631/10	L2		<i>Df(3R)by62</i>	1258/15	E		<i>Df(3R)P14</i>
0528/10	E		<i>Df(3R)by62</i>	0665/08	E		<i>Df(3R)P14</i>
0455/19	L3		<i>Df(3R)by62</i>	0065/17	E		<i>Df(3R)P14</i>
0224/40	E-L1		<i>Df(3R)by62</i>	0592/13	L3		<i>Df(3R)P14</i>
0469/18	L1		<i>Df(3R)by62</i>	1489/07	E		<i>Df(3R)P14</i>
0480/06	L2		<i>Df(3R)by62</i>	0578/06	E		<i>Df(3R)P14</i>
0223/61	L2		<i>Df(3R)by62</i>	0569/16	E		<i>Df(3R)P14</i>
0480/13	pP		<i>Df(3R)by62</i>	0118/32	E-L1		<i>Df(3R)P14</i>
1056/05	L2		<i>Df(3R)by62</i>	0545/16	L3		<i>Df(3R)P14</i>
0232/06	E-L1		<i>Df(3R)by62</i>	0545/15	L3-pP		<i>Df(3R)P14</i>
1115/15	E		<i>Df(3R)by62</i>	0202/02	E		<i>Df(3R)P14</i>

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Line	Lethal phase	Location	Deficiency	Line	Lethal phase	Location	Deficiency
0689/11	E		<i>Df(3R)P14</i>	1305/10	E		<i>Df(3R)sbd26</i>
0696/17	E		<i>Df(3R)sbd26</i>	0458/31	L3-pP		<i>Df(3R)sbd26</i>
1268/01	L2		<i>Df(3R)sbd26</i>	1478/04	P		<i>Df(3R)sbd26</i>
0829/01	P		<i>Df(3R)sbd26</i>	1024/09	P		<i>Df(3R)sbd26</i>
1008/06	P-pA		<i>Df(3R)sbd26</i>	0046/13	pP-P		<i>Df(3R)sbd26</i>
0051/15	E-L1		<i>Df(3R)sbd26</i>	0945/03	E		<i>Df(3R)sbd26</i>
1336/10	P		<i>Df(3R)sbd26</i>	1128/03	E		<i>Df(3R)sbd26</i>
0636/02	E		<i>Df(3R)sbd26</i>	0995/04	pA		<i>Df(3R)sbd26</i>
0633/09	E		<i>Df(3R)sbd26</i>	0716/15	P		<i>Df(3R)sbd26</i>
0598/13	P-pA		<i>Df(3R)sbd26</i>	0826/09	L3		<i>Df(3R)sbd26 and Df(3L)cat</i>
0010/12	E		<i>Df(3R)sbd26</i>	1412/03	E		<i>Df(3R)T61</i>
0495/14	E		<i>Df(3R)sbd26</i>				

APPENDIX B

Probe	Location of rescued fragment	Cosmid contigs identified	P1 clones identified
943/10	86E1-2	2090 = 16B2-37G8- 76H2 -185A11-194C10 168 = 127G1- 128A3 -168F2- 177G3	
979/7	86E10-12	2150 = 24B1- 55F4 2776 = 24H2- 188E6 75G2	DS05232
369/16	86E1-2	2090 = 16B2-37G8- 76H2 -185A11-194C10 168 = 127G1- 128A3 -168F2- 177G3	
95/12	86E1-6	877 = 27D7 -40G7-40H7- 58A5 921 = 42F11 -42G11-44A11-47G11	DS01296
671/2	86E3-6	877 = 27D7 -40G7-40H7- 58A5 2090 = 16B2-37G8- 76H2 -185A11-194C10 921 = 42F11 -42G11-44A11-47G11 2068 = 108D3 -150A2	DS01296
62A1(cDNA)	86E3-6	837 = 10C5 - 28B10 -103H11-159H12 1555 = 13A8- 68E8 -73G1-96G12-128A3 2406 = 10B4- 149G1 96B1	
51A2(cDNA)	86E4-8	837 = 10C5 - 28B10 -103H11-159H12 1555 = 13A8- 68E8 -73G1-96G12-128A3 2406 = 10B4- 149G1 38G6 96B1	
762/1	86E9-11	2150 = 24B1- 55F4 2776 = 24H2- 188E6 16E6 75G2	DS05232
96/47	86E9-12	2150 = 24B1- 55F4 2776 = 24H2- 188E6	DS05232
979/7	86E10-12	2150 = 24B1- 55F4 2776 = 24H2- 188E6	DS05232
300/3	86E14-20	1686 = 132E5 -139H4 2010 = 24C12-85H8-136H10	
TAF30α	86F1-2	2010 = 24C12-85H8-136H10	
1250/12	86F1-3	1397 = 25E11 -37A1-45E2- 87D8 -94H8-121A1- 131E3 - 187C3	DS00360 DS00597 DS08846
440/6	86F3-6	1397 = 25E11 -37A1-45E2- 87D8 -94H8-121A1- 131E3 - 187C3 1429 = 1A8-58A4- 132C4 -144D8-163H4-193C8 1753 = 50E10- 144G9 - 170B9 3E9 66B2 88C6	DS00360 DS00597 DS08846
1132/8	86F3-6	1397 = 25E11 -37A1-45E2- 87D8 -94H8-121A1- 131E3 - 187C3 1429 = 1A8-58A4- 132C4 -144D8-163H4-193C8 1753 = 50E10- 144G9 - 170B9 3E9 88C6	DS00360 DS00597 DS08846
501/16	86F3-6	1397 = 25E11 -37A1-45E2- 87D8 -94H8-121A1- 131E3 - 187C3 1753 = 50E10- 144G9 - 170B9 3E9 88C6	DS00360 DS00597
492/15	86F3-5	2770 = 107F4 -172A5 1429 = 1A8-58A4- 132C4 -144D8-163H4-193C8 1753 = 50E10- 144G9 - 170B9 3E9 66B2 88C6	DS00360 DS00597
493/14	86F1-6	1429 = 1A8-58A4- 132C4 -144D8-163H4-193C8 1753 = 50E10- 144G9 - 170B9 3E9 66B2 88C6	DS00360 DS00597

APPENDIX B

Continued

Probe	Location of rescued fragment	Cosmid contigs identified	P1 clones identified
58/18	86F4-7	1555 = 13A8-68E8-73G1-96G12-128F12 1429 = 1A8-58A4-132C4-144D8-163H4-193C8 1753 = 50E10-144G9-170B9 3E9 66B2 88C6	DS00360 DS00597
504/7	86F4-6	1429 = 1A8-58A4-132C4-144D8-163H4-193C8 1753 = 50E10-144G9-170B9 3E9 66B2 88C6	DS00360 DS00597
Lk6	86F3-6	1397 = 25E11-37A1-45E2-87D8-94H8-121A1-131E3-187C3 3E9	
549/7	87A1-2	1429 = 1A8-58A4-132C4-144D8-163H4-193C8 141 = 3H2-66D7 22F5 32F2	DS00597
110/46	87A1-2	1429 = 1A8-58A4-132C4-144D8-163H4-193C8 22F5 32F2	
1261/13	87A5-8 and 87C1-3	1655 = 91H3-98F5-145H12-147B5-194E3 2909 = 156D9-166B7 2817 = 15A12-101E4 1761 = 140A9-140B9-140A10 1508 = 39F12-137H11-15D12-165H11 4A1 5G6 50G2 129A7 169B5	
1353/1	87A8-B2	1655 = 91H3-98F5-145H12-147B5-194E3 2909 = 156D9-166B7 2817 = 15A12-101E4 12H12 48A5 95A12 118F5 129A7 152H6 153H1 176D11 8E7 58C12	
288/13	87B10-15	1508 = 39F12-137H11-145D12-165H11 3H2	DS03200
842/4	87B10-15	1508 = 39F12-137H11-145D12-165H11 2485 = 3H2-65D1	DS03200
Pp1-87B	87B11-14	2061 = 14E9-36A4-61B2-76B12-103D1-103D2-125B5 1508 = 39F12-137H11-145D12-165H11 181H5	
α - γ element	87C1-2	1761 = 140A9-140B9-140A10 4A1 50G2 169B5 171C11 189A5	
1208/13 516/1	87C1-3 87C1-3	No hybridizing cosmid 1761 = 140A9-140B9-140A10 1718 = 78D3-130H3 2485 = 3H2-65D1 4A1 12H12 48A5 50G2 63E8 66D5 99G4 99G6	DS05532

APPENDIX B

Continued

Probe	Location of rescued fragment	Cosmid contigs identified	P1 clones identified
Arp	87C4-5	134C5 169B5 171C11 189A5 <i>1235 = 1D11-85G4-116B1-147F10</i> <i>854 = 16A2-26E1-134F11-159G11</i> 139A12 191A11	
1465/7	87C5-7	<i>1235 = 1D11-85G4-116B1-147F10</i> <i>854 = 16A2-26E1-134F11-159G11</i> <i>1831 = 85E12-138C2-148E8-194B4</i> <i>1235 = 1D11-85G4-116B1-147F10</i> <i>854 = 16A2-26E1-134F11-159G11</i> <i>1831 = 85E12-138C2-148E8-194B4</i>	DS00573
742/5	87C5-9	<i>1235 = 1D11-85G4-116B1-147F10</i> <i>854 = 16A2-26E1-134F11-159G11</i> <i>1831 = 85E12-138C2-148E8-194B4</i>	DS00573
369/9	87C8-D1	<i>380 = 1E7-12G2</i> <i>854 = 16A2-26E1-134F11-159G11</i> 113F12 139A12	DS08304
30/6	87D1-2	<i>380 = 1E7-12G2</i> <i>854 = 16A2-26E-134F11-159G11</i> 113F12 139A12	DS08304
1250/6 <i>rosy</i>	87D1-5 87D6-10	No hybridizing cosmid <i>2639 = 31E9-35E9-71A3-151H10-153H1</i> 69F12	DS08447
256/16	87D6-14	<i>2639 = 31E9-35E9-71A3-151H10-153H1</i> <i>1624 = 99E7-116A9</i> 69F12 136F9	DS03202
221/22	87D9-14	<i>2639 = 31E9-35E9-71A3-151H10-153H1</i> <i>1624 = 99E7-116A9</i> 69F12 136F9	DS03202
14/7	87D10-14	<i>2639 = 31E9-35E9-71A3-151H10-153H1</i> <i>1624 = 99E7-116A9</i> 69F12 136F9	DS03202
263/16 <i>Ace</i> <i>Act87E</i> 1131/5	87D10-14 87E1-3 87E10-87F1 87E10-F1	No hybridizing cosmid 163E11 84H8 <i>1997 = 43E4-43F4</i> <i>1865 = 83A2-143G10</i> 61A10 167H1 185D8	DS04954
1041/1	87F2-5	<i>1865 = 83A2-143G10</i> 185D8	DS03157
Rbp4	87F3-6	<i>1997 = 43E4-43F4</i> <i>1865 = 83A2-143G10</i> 61A10 185D8	
Hrb87F	87F3-6	<i>1997 = 43E4-43F4</i> <i>1865 = 83A2-143G10</i> 61A10 185D8	
86/14	87F6-10	<i>1997 = 43E4-43F4</i> <i>1865 = 83A2-143G10</i> 167H1 185D8	
299/10	87F9-12	<i>1997 = 43E4-43F4</i> <i>1865 = 83A2-143G10</i> 61A10 167H1	

Italicized numerals represent cosmid contigs. Individual cosmids within contigs that show hybridization to the probe are shown in bold.